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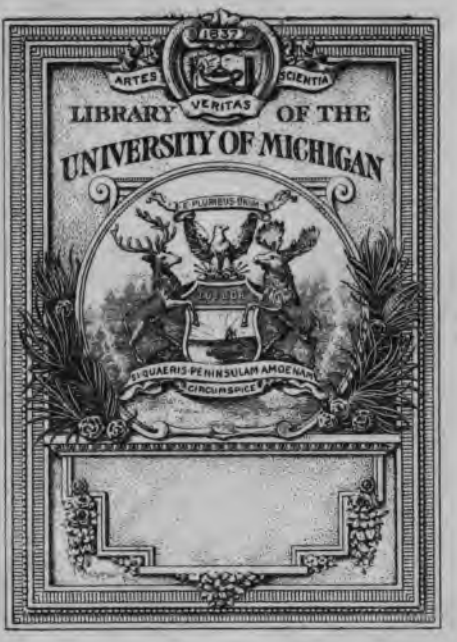
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PUBLISHED BY THE

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VOLUME THE TWENTY-EIGHTH.

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1841. ROBERT WILLIAMS, M.D.
1843. EDWARD STANLEY.
1845. WILLIAM F. CHAMBERS, M.D., K.G.H.

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1821. JOHN COOKE, M.D.

FELLOWS

OF THE

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AUGUST 1845.

Amongst the non-residents, those marked thus (•) are entitled by composition to receive the Transactions.

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1841 *James Abercrombie, M.D., *Cape of Good Hope.*

1842 William Acton, Esq., *Surgeon to the Islington Dispensary;* 46, *Queen Anne-street, Cavendish-square.*

1818 Walter Adam, M.D., *Physician to the Royal Public Dispensary, Edinburgh.*

1818 Thomas Addison, M.D., *Physician to Guy's Hospital;* 24, *New-street, Spring-gardens.* C. 1826. V. P. 1837.

1814 Joseph Ager, M.D., *Great Portland-street.* C. 1836.

1819 *James Ainge, Esq., *Fareham, Hants.*

1837 *Ralph Fawsett Ainsworth, M.D., 104, *King-street, Manchester.*

ELECTED

1819 George F. Albert, Esq.

1839 Rutherford Alcock, Esq., K.C.T., K.T.S.

1826 James Alderson, M.D., F.R.S., *Physician to the General Infirmary, Hull.*

1843 C. J. B. Aldis, M.D., *Physician to the London and Surrey Dispensaries, and Lecturer on Medicine to the Aldersgate and Charlotte-street Schools of Medicine; Old Burlington-street.*

1813 Henry Alexander, Esq., *Surgeon-Oculist in Ordinary to the Queen, and Surgeon to the Royal Infirmary for Diseases of the Eye; 6, Cork-street.* C. 1840.

1836 Henry Ancell, Esq., *Surgeon to the Western General Dispensary; 3, Norfolk-crescent, Oxford-square.*

1817 Alexander Anderson, Esq.

1816 John Goldwyer Andrews, Esq., *Surgeon to the London Hospital; 4, St. Helen's-place.* C. 1836. V. P. 1840.

1820 Thomas F. Andrews, M.D., *Norfolk, Virginia.*

1813 William Ankers, Esq., *Knutsford.*

1819 Professor Antommarchi, *Florence.*

1818 William Withering Arnold, M.D., *Physician to the Infirmary and Lunatic Asylum, Leicester.*

1825 Thomas Graham Arnold, M.D., *Stamford.*

1819 James M. Arnott, Esq., F.R.S., *Surgeon to the Middlesex Hospital; New Burlington-street.* L. 1826. T. 1835. V. P. 1832.

1828 Neil Arnott, M.D., F.R.S., *Physician Extraordinary to the Queen; Bedford-square.* C. 1835.

1817 John Ashburner, M.D., M.R.I.A., *Physician-Accoucheur to the Queen Charlotte's Lying-in Hospital, and Physician-Accoucheur to the Middlesex Hospital; Wimpole-street.* C. 1821.

1822 Samuel Ashwell, M.D., 16, *Grafton-street.* C. 1827.

1841 John Avery, Esq., *Surgeon to the Charing Cross Hospital; 17, Saville-row.*

1825 Benjamin G. Babington, M.D., F.R.S., VICE-PRESIDENT, *Physician to Guy's Hospital, and Physician to the Deaf and Dumb Institution; 31, George-street, Hanover-square.* C. 1829. V. P. 1845.

ELECTED

1819 John Carr Badeley, M.D., *Chelmsford.*

1820 *John H. Badley, Esq., *Dudley.*

1838 Francis Badgley, M.D., *Montreal, Upper Canada.*

1840 William Bainbridge, Esq., *Upper Tooting.*

1836 Andrew Wood Baird, M.D., *Ipswich.*

1816 *William Baker, M.D., *Physician to the Derbyshire General Infirmary; Derby.*

1839 T. Graham Balfour, M.D., *Grenadier Guards, Army and Navy Club, St. James's-square.*

1837 William Baly, M.D., *Physician to the General Penitentiary, Millbank, and Lecturer on Forensic Medicine at St. Bartholomew's Hospital;* 28, *Spring-gardens.* C. 1845.

1833 Alfred Barker, M.D., *Physician to St. Thomas's Hospital;* 15, *Grafton-street, Bond-street.* C. 1844.

1843 Thomas Herbert Barker, Esq., *formerly House-Surgeon to University College Hospital; Priory-terrace, Bedford.*

1843 Christopher Hewetson Barnes, Esq., *late Surgeon Hon. East India Company's Service; Belle-Vue House, Notting-hill.*

1815 *John Baron, M.D., *Cheltenham.*

1840 Benjamin Barrow, Esq., *Liverpool.*

1822 James Bartlett, M.D., *Physician to His Royal Highness the Duke of Cambridge;* 10, *Bentinck-street.*

1844 William R. Basham, M.D., *Physician to the Westminster Hospital; Chester-street, Grosvenor-place.*

1836 William Beaumont, Esq., *Professor of Surgery in the University of King's College; Toronto, Upper Canada.*

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1840 Charles Beevor, Esq., *Surgeon to the St. Marylebone Dispensary;* 49, *Berners-street.*

1818 *Joseph Bell, Esq., *Surgeon to the Royal Infirmary; Edinburgh.*

1819 Thomas Bell, Esq., F.R.S., L.S., and G.S., *Lecturer on Diseases of the Teeth, at Guy's Hospital;* 17, *New Broad-street.* C. 1832.

1845 Edward Unwin Berry, Esq., *James-street, Covent-garden.*

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1818 John Jeremiah Bigsby, M.D., *Newark, Nottinghamshire.*

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1845 Golding Bird, M.D., A.M., *Assistant-Physician to, and Lecturer on Materia Medica at, Guy's Hospital; Myddelton-square.*

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1839 Richard Blagden, Esq., *Surgeon-Accoucheur, and Surgeon Extraordinary, to the Queen; Surgeon in Ordinary to Her Royal Highness the Duchess of Kent; Albemarle-street.*

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1841 James Blake, Esq., 16, *Pall-mall.*

1840 Peyton Blakiston, M.D., F.R.S., *Birmingham.*

1811 *Henry C. Boisragon, M.D., *Cheltenham.*

1823 Louis Henry Bojanus, M.D., *Wilna.*

1816 Hugh Bone, M.D., *Inspector-General of Hospitals.*

1810 John Booth, M.D., *Physician to the General Hospital at Birmingham.*

1841 William Bowman, Esq., F.R.S., *Assistant-Surgeon to King's College Hospital;* 14, *Golden-square.*

1806 John Bostock, M.D., F.R.S., 22, *Upper Bedford-place.* C. 1818. T. 1819. V. P. 1821.

1844 Robert Brandon, Esq.

1814 Richard Bright, M.D., F.R.S., *Physician Extraordinary to the Queen, and Consulting Physician to Guy's Hospital; Saville-row.* C. 1821. V. P. 1827. P. 1837.

1813 Sir Benjamin C. Brodie, Bart., V.P.R.S., *Serjeant-Surgeon to the Queen, Surgeon in Ordinary to His Royal Highness Prince Albert; Saville-row.* C. 1814. V. P. 1816. P. 1839.

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1844 Charles Brooke, Esq., *Keppel-street, Russell-square.*

1842 Charles Blakely Brown, M.D., *Physician-Accoucheur to the St. George's and St. James's Dispensary;* 38, *Hill-street, Berkeley-square.*

1818 *Samuel Barwick Brtice, Esq., *Surgeon to the Forces; Ripon.*

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1844 A. J. Burmester, Esq., 7, *Stanhope-street, Gloucester-gate, Regent's-park.*

1833 George Burrows, M.D., TREASURER, *Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital;* 45, *Queen Anne-street.* C. 1839. T. 1845.

1820 Samuel Burrows, Esq.

1835 Henry Burton, M.D., *Physician to St. Thomas's Hospital;* 41, *Jermyn-street.* C. 1842.

1837 George Busk, Esq., *Hospital-ship Dreadnought; Greenwich.*

1818 John Butter, M.D., F.R.S., F.L.S., *Physician to the Plymouth Eye Infirmary; Plymouth.*

1832 *William Campbell, M.D., *Physician to the New Town Dispensary, and Lecturer on Midwifery; Edinburgh.*

1838 *Alexander Campbell, M.D., *Bombay.*

1842 Henry Cantis, Esq., 8, *Maddox-street, Hanover-square.*

1839 Robert Carswell, M.D., *Physician to their Majesties the King and Queen of the Belgians; Brussels.*

1825 Harry Carter, M.D., *Physician to the Kent and Canterbury Hospital; Canterbury.*

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1820 Samuel Cartwright, Esq., F.R.S., *Burlington-street.*

1845 Samuel Cartwright, Jun., Esq., *Sackville-street, Piccadilly.*

1839 William Cathrow, Esq., *Weymouth-street.*

1845 William Oliver Chalk, Esq., *Nottingham-terrace, New-road.*

1818 Richard Chamberlaine, Esq., *Kingston, Jamaica.*

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1844 Thomas K. Chambers, M.D., *Curzon-street, May-fair*.

1837 Henry T. Chapman, Esq., *Ampthill, Bedfordshire*.

1838 George Chaplin Child, M.D., *Physician to the Westminster General Dispensary*; 27, *Mortimer-street*.

1842 W. D. Chowne, M.D., *Physician to the Charing Cross Hospital*; *Princes-street, Cavendish-square*.

1827 Sir James Clark, Bart., M.D., F.R.S., *Physician to the Queen, Physician in Ordinary to His Royal Highness Prince Albert, and Consulting Physician to their Majesties the King and Queen of the Belgians*; *Brook-street*. C. 1830. V. P. 1832.

1839 F. Le Gros Clark, Esq., *Assistant-Surgeon to, and Lecturer on Descriptive and Surgical Anatomy at, St. Thomas's Hospital*; 30, *Finsbury-square*.

1845 John Clark, M.D., *Staff Surgeon 2nd class*; 12, *Beaumont-street, Portland-place*.

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1842 Oscar M. P. Clayton, Esq., 3, *Percy-street, Bedford-square*.

1827 John Clendinning, M.D., F.R.S., *Physician to the St. Marylebone Infirmary*; 16, *Wimpole-street*. C. 1832. S. 1834. V. P. 1840.

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1818 Robert Cole, Esq., F.L.S., *Holybourne, Hampshire*.

1828 John Conolly, M.D., *Hanwell*.

1839 John C. Cooke, M.D., *Coventry*.

1840 *William Robert Cooke, Esq., *Burford, Oxfordshire*.

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1819 George Cooper, Esq., *Brentford*.

1820 Benjamin Cooper, Esq., *Stamford*.

1843 William W. Cooper, Esq., *Senior Surgeon to the North London Ophthalmic Institution and to the Honourable Artillery Company*; 2, *Tenterden-street, Hanover-square*.

1841 George Lewis Cooper, Esq., *Surgeon to the Bloomsbury Dispensary*; 35, *Keppel-street, Russell-square*.

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1841 Holmes Coote, Esq., *Surgeon to the North London Ophthalmic Institution; 24, Argyll-street.*

1835 George F. Copeland, Esq., *Cheltenham.*

1822 James Copland, M.D., F.R.S., *Consulting Physician to Queen Charlotte's Lying-in Hospital; 5, Old Burlington-street.* C. 1830. V. P. 1838.

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1814 *William Cother, Esq., *Surgeon to the Infirmary, Gloucester.*

1828 William Coulson, Esq., *Surgeon to the Magdalen Hospital, Consulting Surgeon to the City Lying-in Hospital; Frederick's-place, Old Jewry.* C. 1831. L. 1832.

1836 William Travers Cox, M.D., 2, *Stanhope-place, Hyde-park.*

1817 Sir Philip Crampton, Bart., F.R.S., *Surgeon-General to the Forces in Ireland; Dublin.*

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1841 M. A. N. Crawford, M.D., *Physician to the Middlesex Hospital; 62, Upper Berkeley-street, Portman-square.*

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1836 George Cursham, M.D., SECRETARY, *Physician to the Asylum for Female Orphans; 5, Saville-row.* S. 1842.

1822 Christopher John Cusack, Esq.

1828 Adolphe Dalmas, M.D., *Paris.*

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1836 *James S. Daniel, Esq., *Ramsgate.*

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1818 Henry Davies, M.D., *Physician to the British Lying-in Hospital, Brownlow-street; Saville-row.* C. 1827.

1817 Thomas Davis, Esq., *Hampstead.* C. 1837.

1820 Thomas Davis, Esq., *Brook-street, Hanover-square.* C. 1843.

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1839 James Dixon, Esq., *Assistant-Surgeon to the Royal London Ophthalmic Hospital, and Demonstrator of Anatomy at St. Thomas's Hospital;* 37, *Broad-street-buildings.*

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1845 John Dodd, Esq,, *Portman-street, Portman-square.*

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1839 Henry Pye Lewis Drew, Esq., *Torrington-square.*

1843 Thomas Jones Drury, M.D., *Physician to the Salop Infirmary; Shrewsbury.*

1845 George Duff, M.D., 14, *Gloucester-road, Hyde-park-square.*

1845 Edward W. Duffin, Esq., *Langham-place, Portland-place.*

1833 William Dunbar, M.D., *Bombay.*

1833 Robert Dunn, Esq., *Norfolk-street, Strand.* C. 1845.

1843 C. M. Durrant, M.D., *Physician to the East Suffolk and Ipswich Hospital; Ipswich.*

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1824 George Edwards, Esq.

1823 C. C. Egerton, Esq., *India.*

1814 Philip Elliot, M.D., *Bath.*

1838 Thomas Elliotson, M.D., *Physician to the Surrey Dispensary; Clapham.*

1835 William England, M.D., *Wisbeach.*

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1842 John E. Erichsen, Esq., *Lecturer on General Anatomy and Physiology at the Westminster Hospital;* 48, *Welbeck-street, Cavendish-square.*

1815 G. F. D. Evans, M.D. C. 1838.

1836 George F. Evans, M.B., *Physician to the Birmingham Hospital.*

1845 William Julian Evans, M.D., 25, *Henrietta-street, Cavendish-square.*

1841 Sir James Eyre, M.D., 11, *Brook-street, Grosvenor-square.*

1841 Alexander Farquhar, Esq., A.M.

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1831 Robert Ferguson, M.D., *Physician-Accoucheur to the Queen, Physician to the Westminster Lying-in Hospital; Queen-street, May-fair.* C. 1839.

1814 William Fergusson, M.D., *Inspector of Hospitals; Windsor.*

1841 William Fergusson, Esq., *Professor of Surgery in King's College, London; and Surgeon to King's College Hospital;* 8, *Dover-street, Piccadilly.*

1841 William Finch, M.D., F.L.S., *Laverstock-hall, Salisbury.*

1836 John W. Fisher, Esq., *Surgeon-in-Chief to the Metropolitan Police Force;* 21, *Argyll-street.* C. 1843.

1839 G. Lionel Fitzmaurice, Esq., 97, *Gloucester-place, Portman-square.*

1840 Valentine Flood, M.D., *Beaufort-buildings, Strand.*

1842 Thomas Bell Elcock Fletcher, M.D., *Physician to the General Dispensary, Birmingham.*

1841 John Forbes, M.D., F.R.S,, *Physician to Her Majesty's Household; Old Burlington-street.*

1817 *Robert T. Forster, Esq., *Southwell.*

1820 Thomas Forster, M.D., *Hartfield-lodge, East Grinsted.*

1816 John W. Francis, M.D., *Professor of Materia Medica in the University of New York.*

1841 J. Ch. August. Franz, M.D., *Royal German Spa, Brighton.*

1843 Patrick Fraser, M.D., *Assistant-Physician to the London Hospital; Guilford-street, Russell-square.*

1836 John G. French, Esq., *Surgeon to St. James's Infirmary;* 41, *Great Marlborough-street.*

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1815 *George Frederick Furnival, Esq., *Egham.*

1819 John Samuel Gaskoin, Esq., 32, *Clarges-street.* C. 1836.

1819 Henry Gaulter, Esq.

1821 *Richard Francis George, Esq., *Surgeon to the Bath Hospital.*

1841 J. D. George, Esq., F.G.S., *Lecturer on Dental Surgery to University College, and Dental Surgeon to University College Hospital;* 32, *Old Burlington-street.*

1812 George Goldie, M.D., *York.*

1837 Richard H. Golden, M.D., *Physician to the Hospital-ship Dreadnought, Assistant-Physician to St. Thomas's Hospital; John-street, Adelphi.*

1818 James Alexander Gordon, M.D., F.R.S., *Physician to the London Hospital; Lower Grosvenor-street.* S. 1823. C. 1828. V. P. 1829.

1827 R. D. Grainger, Esq., *Lecturer on Anatomy; Anerley, Norwood.* C. 1829. V. P. 1843.

1844 John Grantham, Esq., *Crayford, Kent.*

1816 Joseph H. Green, Esq., F.R.S., *Surgeon to St. Thomas's Hospital; Hadley, Middlesex.* C. 1820. V. P. 1830.

1841 George Gregory, M.D., *Physician to the Small-Pox Hospital;* 31, *Weymouth-street.* S. 1825.

1843 Robert Greenhalgh, Esq., *Upper Charlotte-street, Fitzroy-square.*

1835 William Griffith, Esq., *Surgeon to the Royal Maternity Charity, and Lecturer on Midwifery at the Westminster Hospital; Lower Belgrave-street, Belgrave-square.*

1814 John Grove, M.D., *Salisbury.*

1837 James Manby Gully, M.D., *Holyrood-house, Great Malvern.*

1819 John Gunning, Esq., *Inspector of Hospitals, Paris.*

1841 Charles W. G. Guthrie, Esq., *Saville-row.*

1827 Marshall Hall, M.D., F.R.S., *Manchester-square.* L. 1828. C. 1840. V. P. 1843.

1842 *George Hall, M.D., 14, *Old Steine, Brighton.*

1845 John Hall, M.D., *Staff Surgeon, Junior United Service Club.*

1819 Thomas Hammerton, Esq., 111, *Piccadilly.*

1838 Henry Hancock, Esq., *Surgeon to the Charing Cross Hospital; Harley-street.*

ELECTED

1836 J. F. Harding, Esq., *Spencer-street, Northampton-square.*

1843 Thomas Sunderland Harrison, M.D., F.L.S., *Senior Physician to the Farringdon Dispensary; 5, Little Argyll-street, and Garston Lodge, Somersetshire.*

1841 William Harvey, Esq., *Surgeon to the Freemasons' Female Charity; 43, Great Queen-street, Lincoln's-inn-fields.*

1845 John Havers, Esq., *York-road, Lambeth.*

1816 *John Haviland, M.D., *Regius Professor of Physic in the University of Cambridge; Physician to Addenbrooke's Hospital.*

1828 Cæsar H. Hawkins, Esq., TREASURER, *Surgeon to St. George's Hospital, and Lecturer on Surgery; 26, Lower Grosvenor-street.* C. 1830. V. P. 1838. T. 1841.

1838 Charles Hawkins, Esq., 2, *Court-yard, Albany.*

1820 Thomas Emerson Headlam, M.D., *Newcastle-upon-Tyne.*

1829 T. Heberden, M.D., 11, *Upper Brook-street.*

1844 John Hennen, M.D., 24, *Upper Southwick-street, Hyde-park.*

1821 Vincent Herberski, M.D., *Professor of Medicine in the University of Wilna.*

1843 Prescott Gardiner Hewett, Esq., *Curator of St. George's Hospital Museum; 4, Vigo-street, Burlington-gardens.*

1841 *Nathaniel Highmore, Esq., *Consulting Surgeon to the Weymouth and Dorsetshire Eye Infirmary; Sherborne.*

1814 *William Hill, Esq., *Wootton-under-Edge.*

1842 William Augustus Hillman, Esq., *Argyll-street.*

1841 John Hilton, Esq., F.R.S., *Assistant-Surgeon to Guy's Hospital; 10, New Broad-street.*

1840 Thomas Hodgkin, M.D., 9, *Brook-street.* C. 1842.

1813 Joseph Hodgson, Esq., F.R.S., *Surgeon to the General Hospital, and to the Eye Infirmary, Birmingham.* C. 1817.

1835 T. H. Holberton, Esq., *Surgeon Extraordinary to the Queen Dowager; Hampton.*

1843 Luther Holden, Esq., *Old Jewry.*

1814 Henry Holland, M.D., F.R.S., *Physician Extraordinary to the Queen, and Physician in Ordinary to His Royal Highness Prince Albert; 25, Brook-street.* C. 1817. V. P. 1826.

ELECTED

1819 *John Howell, M.D., F.R.S. Ed., *Clifton.*

1828 *Edward Howell, M.D., *Swansea.*

1844 Edwin Humby, Esq., *Windsor-terrace, Maida-hill.*

1822 Robert Hume, M.D., *Inspector of Hospitals;* 9, *Curzon-street.* V. P. 1836.

1840 Henry Hunt, M.D., *Brook-street, Hanover-square.*

1842 Christopher Hunter, Esq., *Downham, Norfolk.*

1820 William Hutchinson, M.D.

1840 Charles Hutton, M.D., 25, *Motcomb-street, Belgrave-square.*

1838 William Ifil, M.D.

1826 William Ingram, Esq., *Midhurst.*

1839 A. R. Jackson, M.D., *East India Company's Depôt, Warley Barracks, Essex.*

1845 *Henry Jackson, Esq., *Surgeon to the Sheffield General Infirmary; St. James's-row, Sheffield.*

1841 Paul Jackson, Esq., *Thayer-street, Manchester-square.*

1841 Maximilian M. Jacobovicz, M.D., *Pesth.*

1825 John B. James, M.D.

1844 Samuel John Jeaffreson, M.D., *Physician to the Chelsea, Belgrave and Brompton Dispensary;* 22, *Half-moon-street.*

1839 Julius Jeffreys, Esq., F.R.S., 25 A, *Norfolk-crescent, Hyde-park.*

1840 *G. Samuel Jenks, M.D., *Brighton.*

1821 Edward Johnson, M.D., *Weymouth.*

1837 H. C. Johnson, Esq., *Assistant-Surgeon to St. George's Hospital, and Lecturer on Descriptive and Surgical Anatomy in the St. George's Hospital Medical School;* 6, *Saville-row.*

1844 John Johnston, Esq., 2, *Stafford-street, Bond-street.*

1835 H. D. Jones, Esq., 20, *Soho-square.*

1844 Henry Bence Jones, M.D., *Grosvenor-street.*

1837 T. W. Jones, M.D., 19, *Finsbury-pavement, Finsbury-square.*

1829 *G. Julius, Esq., *Richmond.*

1816 *George Hermann Kauffmann, M.D., *Hanover.*

1815 Robert Keate, Esq., *Serjeant-Surgeon to the Queen, Surgeon to Her Royal Highness the Duchess of Gloucester, and*

ELECTED

Surgeon to St. George's Hospital; 15, *Albemarle-street.* C. 1818. V. P. 1826.

1822 Robert Masters Kerrison, M.D., F.R.S., 12, *New Burlington-street.* C. 1840.

1838 L. P. Kell, M.D., *Bridge-street, Westminster.*

1839 *David King, M.D., *Eltham.*

1836 P. N. Kingston, M.D., *Physician to the Westminster Hospital; Curzon-street, May-fair.*

1806 James Laird, M.D.

1805 William Lambe, M.D., *Penwood Wedly, Herefordshire.* C. 1807.

1823 Edmund Lambert, M.D., *Salisbury.*

1840 Samuel Lane, Esq., *Assistant-Surgeon to the Lock Hospital, Grosvenor-place.*

1841 *Charles Lashmar, M.D., *Croydon, Surrey.*

1816 G. E. Lawrence, Esq.

1809 William Lawrence, Esq., F.R.S., *Surgeon Extraordinary to the Queen; Surgeon to St. Bartholomew's Hospital, and to Bridewell and Bethlem Hospitals; Lecturer on Surgery at St. Bartholomew's Hospital;* 18, *White-hall-place.* S. 1813. V. P. 1818. C. 1820. T. 1821. P. 1831.

1840 Thomas Laycock, M.D., *York.*

*Jesse Leach, Esq., *Heywood, near Bury, Lancashire.*

1823 John G. Leath, M.D.

1822 John Joseph Ledsam, Esq., *Surgeon to the Birmingham Eye Infirmary.*

1822 Robert Lee, M.D., F.R.S., *Physician to the British Lying-in Hospital, and Physician-Accoucheur to the St. Marylebone Infirmary; Lecturer on Midwifery at St. George's Hospital;* 4, *Saville-row.* C. 1829. S. 1830. V. P. 1835.

1823 Henry Lee, M.D., 20, *Keppel-street, Russell-square.* C. 1837. S. 1839.

1842 Edwin Lee, Esq., 170, *North-street, Brighton.*

1843 Henry Lee, Esq., 13, *Dover-street, Piccadilly.*

1843 John Leeson, Esq., 4, *Finsbury-square.*

1843 Sir George Lefevre, M.D., *Brook-street, Grosvenor-square.*

ELECTED

1836 Frederick Leighton, M.D.

1806 John Lind, M.D.

1835 Robert Liston, Esq., F.R.S., *Surgeon to University College Hospital; 5, Clifford-street, Bond-street.* C. 1840.

1818 Robert Lloyd, M.D.

1824 Eusebius Arthur Lloyd, Esq., *Assistant-Surgeon to St. Bartholomew's Hospital, and Surgeon to Christ's Hospital;* 14, *Bedford-row.* S. 1827. V. P. 1838. C. 1843.

1820 J. G. Locher, M.C.D., *Town Physician of Zurich.*

1824 Charles Locock, M.D., *First Physician-Accoucheur to the Queen; Physician to the Queen Dowager, and to the Westminster Lying-in Hospital; Hertford-street, May-fair.* C. 1826. V. P. 1841.

1844 Edward Lonsdale, Esq., *Assistant-Surgeon to the Orthopædic Institution; Guildford-street, Russell-square.*

1836 Joseph S. Löwenfeld, M.D., *Berbice.*

1815 *Peter Luard, M.D.

1814 Sir James Macgrigor, Bart., M.D., F.R.S. L. and Ed., *Director-General of the Medical Department of the Army; Camden-hill, Kensington.* C. 1820. V. P. 1815.

1823 George Macilwain, Esq., *Consulting Surgeon to the Finsbury Dispensary; Court-yard, Albany.* C. 1829.

1818 W. Mackenzie, Esq., *Surgeon to the Eye Infirmary, Glasgow.*

1822 Richard Macintosh, M.D.

1844 James Sutherland Mackintosh, Esq., *Wilton-place, Knightsbridge.*

1839 William Macintyre, M.D., *Harley-street.*

1844 Daniel Maclachlan, M.D., *Physician to the Royal Hospital, Chelsea.*

1842 John Macnaught, M.D., *Bedford-street, Liverpool.*

1835 D. C. Macreight, M.D., *St. Hillier's, Jersey.*

1837 A. M. M'Whinnie, Esq., *Assistant-Teacher of Practical Anatomy at St. Bartholomew's Hospital; Bridge-street, Blackfriars.*

1836 John Malyn, Esq., *Surgeon to the Western Dispensary, and to the Infirmary of St. Margaret and St. John;* 12, *James-street, Buckingham-gate.*

ELECTED

1824 Sir Henry Marsh, Bart., M.D., *Dublin.*

1838 Thomas Parr Marsh, M.D., *Physician to the Salop Infirmary, Shrewsbury.*

1840 John Marston, Esq., 6, *Devonshire-street, Portland-place.*

1841 James Ranald Martin, Esq., F.R.S., 71A, *Grosvenor-street.*

1819 *John Masfen, Esq., *Surgeon to the County General Infirmary, and Fever Hospital, Stafford.*

1818 J. P. Maunoir, *Professor of Surgery at Geneva.*

1820 Herbert Mayo, Esq., F.R.S. S. 1825. V. P. 1834.

1837 Thomas Mayo, M.D., F.R.S., *Physician to the St. Marylebone Infirmary; Wimpole-street.* S. 1841.

1839 R. H. Meade, Esq., *Bradford, Yorkshire.*

1819 *Thomas Medhurst, Esq., *Hurstbourne Tarrant.*

1811 Samuel Merriman, M.D., F.L.S., 34, *Brook-sreet.* C. 1813. V. P. 1827. T. 1837.

1837 S. W. J. Merriman, M.D., *Physician to the Westminster General Dispensary; Brook-street.*

1815 Augustus Meyer, M.D., *St. Petersburgh.*

1840 Richard Middlemore, Esq., *Surgeon to the Eye Infirmary, Birmingham,*

1818 *Patrick Miller, M.D., F.R.S. ED., *Physician to the Devon and Exeter Hospitals, and to the Lunatic Asylum; Exeter.*

1844 Nathaniel Montefiore, Esq., 4, *Great Stanhope-street, Mayfair.*

1828 Joseph Moore, M.D., *Physician to the Royal Freemasons' Female Charity;* 10, *Saville-row.* C. 1837.

1836 George Moore, Esq., M.D., *Hastings.*

1842 Thomas Morton, Esq., *Assistant-Surgeon to University College Hospital, and Surgeon to the Queen's Prison;* 7, *Woburn-place, Russell-square.*

1814 *George Frederick Mühry, M.D., *Hanover.*

1841 Edward William Murphy, M.D., *Professor of Midwifery in Universit y College; Henrietta-street, Cavendish-square.*

1845 Thomas D. Mutter, M.D., *Professor of Surgery in Jefferson Medical College, Philadelphia.*

ELECTED

1840 Robert Nairne, M.D., *Physician to St. George's Hospital; 44, Charles-street, Berkeley-square.*

1831 Alexander Nasmyth, Esq., *Surgeon-Dentist to His Royal Highness Prince Albert;* 13, *George-street, Hanover-square.* C. 1844.

1805 Thomas Nelson, M.D., *Tonbridge Wells.* C. 1810. V. P. 1836.

1835 Thomas Andrew Nelson, M.D, 41, *George-street, Portman-square.*

1843 Edward Newton, Esq., *Howland-street, Fitzroy-square.*

1816 Thomas Nixon, Esq., *Surgeon-Major to the First Regiment of Foot Guards.*

1819 *George Norman, Esq., *Surgeon to the United Hospital and Puerperal Charity; Bath.*

1845 Henry Norris, Esq., *South Petherton, Somerset.*

1829 John North, Esq., *Lecturer on Midwifery at the Middlesex Hospital;* 9, *Gloucester-place.* C. 1835.

1843 William O'Connor, Esq., 69, *George-street, Portman-square.*

1822 James Ady Ogle, M.D., F.R.S., *Clinical and Aldrichian Professor of Medicine, Oxford; and Senior Physician to the Radcliffe Infirmary.*

1844 Edward Lodge Ogle, Esq., 25, *South-street, Grosvenor-square.*

1842 William P. Ormerod, Esq., *Demonstrator of Anatomy at St. Bartholomew's Hospital.*

1844 Drewry Ottley, Esq., *Bedford-place, Russell-square.*

1840 James Paget, Esq., *Lecturer on General and Morbid Anatomy and Physiology, and Warden of the Collegiate Establishment at St. Bartholomew's Hospital.*

1806 *Robert Paley, M.D., *Bishopston Grange, near Ripon, Yorkshire.*

1837 George Pardoe, M.D., *Cavendish-square.*

1814 John Ranicar Park, M.D.

1836 J. W. Langston Parker, Esq., *Birmingham.*

1843 *Charles Lewis Parker, Esq., A.M., *Surgeon to the Radcliffe Infirmary, Oxford.*

1841 John Parkin, Esq., *York-place, Battersea.*

ELECTED

1828 Richard Partridge, Esq., F.R.S., *Surgeon to King's College Hospital, and Professor of Anatomy in King's College, London;* 17, *New-street, Spring-gardens.* S. 1832. C. 1837.

1819 Granville Sharp Pattison, Esq., *New York.*

1845 Thomas Bevill Peacock, M.D., *Physician to the Royal General Dispensary;* 2, *South-place, Finsbury-square.*

1830 Charles P. Pelechin, M.D., *St. Petersburgh.*

1830 William Pennington, Esq., 21, *Montague-place, Russell-square.*

1819 John Pryor Peregrine, Esq., 3, *Half-moon-street.*

1839 Thomas Peregrine, Esq., *Half-moon-street.*

1831 Jonathan Pereira, M.D., F.R.S., F.L.S., *Assistant-Physician to, and Lecturer on Materia Medica at, the London Hospital; Finsbury-square.* C. 1844.

1836 John G. Perry, Esq., VICE-PRESIDENT, *Inspector of Prisons;* 16, *Carlton-house-terrace.* C. 1831. S. 1837. V.P. 1845.

1844 William V. Pettigrew, Esq., 30, *Chester-street, Grosvenor-place.*

1814 *Edward Phillips, M.D., *Physician to the County Hospital; Winchester.*

1837 Benjamin Phillips, Esq., F.R.S., LIBRARIAN, *Assistant-Surgeon to the Westminster Hospital;* 17, *Wimpole-street.* L. 1841.

1836 Isaac Pidduck, M.D., 22, *Montague-street, Russell-square.*

1830 Richard Pinckard, M.D., 18, *Bloomsbury-square.* C. 1836.

1841 Alfred Pitman, M.D., *Montague-place, Russell-square.*

1843 George David Pollock, Esq., 18, *Sackville-street.*

1843 Charles Pope, M.D., M.A., F.L.S., *Temple Cloud, near Bristol.*

1844 John Philips Potter, Esq., *Demonstrator of Anatomy at University College Hospital;* 308, *Regent-street, Langham-place.*

1840 Lewis Powell, Esq., *John-street, Berkeley-square.*

1842 James Powell, M.B., *Great Coram-street, Brunswick-square.*

1839 John Propert, Esq., *New Cavendish-street.*

1814 William Prout, M.D., F.R.S., 40, *Sackville-street.* C. 1816. V. P. 1823.

1843 John Pyle, Esq., *Oxford-terrace, Hyde-park.*

ELECTED

1816 Sir William Pym, M.D., *Inspector of Hospitals.*

1830 Jones Quain, M.D., *Paris.*

1835 Richard Quain, Esq., F.R.S., *Surgeon to University College Hospital, and Professor of Anatomy in the same College; Keppel-street.* C. 1838.

1807 John Ramsey, M.D., *Physician to the Infirmary at Newcastle.*

1821 Henry Reeder, M.D., *Ridge House, Chipping, Sudbury.*

1835 G. Regnoli, *Professor of Surgery in the University of Pisa.*

1842 David Boswell Reid, M.D., *House of Commons.*

1829 John Richardson, M.D., F.R.S., *Surgeon to the Naval Hospital, Chatham.*

1843 Joseph Ridge, M.D., *Cavendish-square.*

1845 Benjamin Ridge, M.D., *Putney, Surrey.*

1817 *John Robb, M.D., *Deputy Inspector of Hospitals.*

1821 Charles Julius Roberts, M.D., *Physician to the Adult Deaf and Dumb and Welsh Charity;* 31, *New Bridge-street.* C. 1827.

1829 *Archibald Robertson, M.D., F.R.S., L. and Ed., *Physician to the General Infirmary, Northampton.*

1843 George Robinson, M.D., 40, *Blackett-street, Newcastle-on-Tyne.*

1845 J. M. Edward Roche, M.D., 12, *Cumberland-street, Portman-square.*

1843 William Roden, M.D., A.M., F.L.S., *Kidderminster.*

1835 G. H. Roe, M.D., *Physician to the Westminster Hospital;* 6, *Hanover-square.* C. 1841.

1836 Arnold Rogers, Esq., 296, *Regent-street.*

1819 Henry S. Roots, M.D., 2, *Russell-square.* C. 1833. V. P. 1834.

1829 Sudlow Roots, Esq., *Kingston-on-Thames.*

1836 Richard Roscoe, M.D., 16, *Milman-street, Bedford-row.*

1835 *Caleb B. Rose, Esq., *Swaffham.*

1845 Henry Mortimer Rowden, Esq., *Lecturer on Anatomy at the Middlesex Hospital School of Medicine; Bayham-terrace, Camden-town.*

1841 Richard Rowland, M.D., *Physician to the Bloomsbury Dispensary;* 7, *Woburn-place, Russell-square,*

ELECTED

1837 J. Forbes Royle, M.D., F.R.S., F.L.S., F.Z.S., *Professor of Materia Medica and Therapeutics in King's College, London; 4, Bulstrode-street, Manchester-square.*

1836 James Russell, Esq., *Birmingham.*

1845 James Russell, Jun., Esq., *Birmingham.*

1843 James Russell, Esq., *Broad-street, Golden-square.*

1827 *Thomas Salter, Esq., F.L.S., *Poole.*

1844 *Thomas Bell Salter, M.D., F.L.S., *Ryde, Isle of Wight.*

1842 George Sampson, Esq., 12, *Chester-street, Belgrave-square.*

1845 Edwin Saunders, Esq., *Surgeon-Dentist and Lecturer on Diseases of the Teeth at St. Thomas's Hospital; Argyll-street.*

1834 Ludwig V. Sauvan, M.D., *Warsaw.*

1840 Augustin Sayer, M.D., 28, *Upper Seymour-street.*

1821 Page Nichol Scott, Esq., *Norwich.*

1824 Edward J. Seymour, M.D., F.R.S., *Physician to St. George's Hospital; Charles-street, Berkeley-square.* C. 1826. S. 1827. V. P. 1830.

1840 William Sharp, Esq., F.R.S., F.G.S., F.R.A.S., *late Senior-Surgeon to the Bradford Infirmary; Humber Bank House, Hull.*

1837 William Sharpey, M.D., F.R.S. L. and Ed., *Professor of Anatomy and Physiology in University College, London; Gloucester-crescent, Regent's-park.*

1836 Alexander Shaw, Esq., *Surgeon to the Middlesex Hospital; Henrietta-street, Cavendish-square.* C. 1842. S. 1843.

1818 Thomas Short, M.D., *Physician to the Forces; Edinburgh.*

1839 Thos. H. Silvester, M.D., *High-street, Clapham.*

1842 John Simon, Esq., F.R.S., *Assistant-Surgeon to King's College Hospital, and Demonstrator of Anatomy in King's College; 11, Wellington-street, Strand.*

1821 Charles Skene, M.D., *Professor of Anatomy and Surgery; Marischal College, Aberdeen.*

1827 George Skene, Esq., *Bedford.*

1812 Joseph Skey, M.D., *Inspector-General of Hospitals.*

1824 Frederick C. Skey, Esq., F.R S., *Assistant-Surgeon to St. Bartholomew's Hospital; Surgeon to the Northern Dispensary; and Lecturer on Anatomy and Surgery at the*

ELECTED

Aldersgate-street Medical School; Grosvenor-street. C. 1828. L. 1829. V. P. 1841.

1810 Noel Thomas Smith, M.D., *Newcastle.*

1822 Southwood Smith, M.D., *Physician to the Fever Hospital, and to the Eastern Dispensary;* 38, *Finsbury-square.* C. 1838.

1835 J. G. Smith, Esq., *late Lecturer on Anatomy and Physiology; Harewood, near Leeds.*

1838 Henry Smith, Esq., *Surgeon to the General Dispensary, Aldersgate-street;* 17, *Henrietta-street, Cavendish-square.*

1845 William Smith, Esq., *Upper Berkeley-place, Bristol.*

1843 Robert William Smith, A.M., M.D., M.R.I.A., *Lecturer on Surgery at the Richmond Hospital School of Medicine; Surgeon to the Talbot General Dispensary and Island Bridge Lunatic Asylum;* 62, *Eccles-street, Dublin.*

1843 John Snow, M.D., *Frith-street, Soho-square.*

1819 *George Snowden, Esq., *Ramsgate.*

1816 *John Smith Soden, Esq., *Surgeon to the United Hospital, to the Eye Infirmary, and to the Penitentiary and Lock Hospital; Bath.*

1830 S. Solly, Esq., F.R.S., *Assistant-Surgeon to St. Thomas's Hospital; Surgeon to the General Dispensary, Aldersgate-street;* 1, *St. Helen's-place.* L. 1838. C. 1845.

1844 Frederick R. Spackman, M.B., *Harpenden, St. Alban's.*

1834 James Spark, Esq., *Newcastle.*

1843 *Stephen Spranger, Esq., *Swatheling House, Southampton.*

1838 G. J. Squibb, Esq., 6, *Orchard-street.*

1835 Richard A. Stafford, Esq., *Surgeon Extraordinary to His Royal Highness the Duke of Cambridge; Surgeon to the St. Marylebone Infirmary; Old Burlington-street.* C. 1840.

1815 Edward Stanley, F.R.S., *Surgeon to St. Bartholomew's Hospital;* 23 A, *Brook-street.* C. 1821. S. 1824. V. P. 1827. P. 1843.

1835 Leonard Stewart, M.D., *Keppel-street.*

1842 Alexander Patrick Stewart, M.D., *Physician to the St. Pancras Dispensary;* 130, *Mount-street, Berkeley-square.*

ELECTED

1839 Thomas Stone, M.D.,

1843 Robert Reeve Storks, Esq., *Gower-street, Bedford-square.*

1844 John Soper Streeter, Esq., *Harpur-street, Red Lion-square.*

1827 William Stroud, M.D., 20, *Great Coram-street.* C. 1831.

1839 Alexander John Sutherland, M.D., *Physician to St. Luke's Hospital; Fludyer-street.*

1842 James Syme, Esq., *Professor of Clinical Surgery in the University of Edinburgh; Charlotte-square, Edinburgh.*

1844 R. W. Tamplin Esq., *Surgeon to the Orthopœdic Institution, Great Queen-street, Lincoln's-inn-fields.*

1840 Thomas Tatum, Esq., *Surgeon to St. George's Hospital, and Lecturer on Anatomy;* 3, *George-street, Hanover-square.*

1835 J. C. Taunton, Esq., *Surgeon to the City of London Truss Society, and to the City Dispensary;* 48, *Hatton-garden.* C. 1840.

1845 John Taylor, M.D., *Professor of Clinical Medicine in University College, and Physician to University College Hospital; Keppel-street, Russell-square.*

1845 Thomas Taylor, Esq., *New Bridge-street, Blackfriars.*

1817 Frederick Thackeray, M.D., *Physician to Addenbrooke's Hospital, Cambridge.*

1805 Honoratus Leigh Thomas, Esq., F.R.S. C. 1806. V.P.1815.

1845 Evan Thomas, Esq., *Pwllheli, North Wales.*

1839 Seth Thompson, M.D., *Assistant-Physician to the Middesex Hospital;* 19, *Brook-street.*

1842 Theophilus Thompson, M.D., *Physician to the Northern Dispensary, and to the Hospital for Consumption and Diseases of the Chest;* 3, *Bedford-square.*

1835 F. Hale Thomson, Esq., *Surgeon to the Westminster Hospital; Berners-street.*

1815 *John Thomson, M.D., F.R.S. Ed., *Surgeon to the Forces; Edinburgh.*

1819 John Thomson, M.D., F.L.S., *Physician to the Finsbury Dispensary;* 80, *Coleman-street.* C. 1833. L. 1834.

1836 John Thurnam, M.D., *The Retreat, York.*

ELECTED

1813 Sir Matthew John Tierney, Bart., 26, *Bruton-street.* C. 1824.

1834 R. B. Todd, M.D., F.R.S., LIBRARIAN, *Physician to King's College Hospital; Professor of Physiology and of General and Morbid Anatomy in King's College;* 26, *Parliament-street.* L. 1842.

1828 James Torrie, M.D., *Aberdeen.*

1843 Joseph Toynbee, Esq., F.R.S., *Surgeon to the St. George's and St. James's Dispensary; Argyll-place, Regent-street.*

1808 Benjamin Travers, Esq., F.R.S., *Surgeon Extraordinary to the Queen; Surgeon in Ordinary to His Royal Highness Prince Albert;* 12, *Bruton-street.* C. 1810. V. P. 1817. P. 1827.

1821 *William Travers, M.D., *Scarborough.*

1841 Matthew Truman, M.D., 44, *Gloucester-place, Kentish-town.*

1835 John Cusson Turner, M.D., *Hanwell-park, Middlesex.*

1845 Thomas Turner, Esq., *Surgeon to the Royal Manchester Infirmary, and Lecturer on Anatomy; Moseley-street, Manchester.*

1843 William Twining, M.D., *Physician to the North London Ophthalmic Institution; Bedford-place, Russell-square.*

1819 Barnard Van Oven, Esq., *Consulting Surgeon to the Charity for delivering Jewish Lying-in Women;* 30, *Gower-street, Bedford-square.*

1845 R. A. Varicas, Esq., 29, *Woburn-place, Russell-square.*

1806 Bowyer Vaux, Esq., *Surgeon to the General Hospital, Birmingham.*

1839 W. R. Vickers, Esq., 32, *Baker-street.*

1814 John P. Vincent, Esq., *Surgeon to St. Bartholomew's Hospital;* 16, *Lincoln's-inn-fields.* C. 1823. V. P. 1837.

1810 James Vose, M.D.

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1841 Robert Wade, Esq., *Surgeon to the Westminster General Dispensary;* 68, *Dean-street.*

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[It is particularly requested that any change of Title or Residence may be communicated to the Secretaries before the 1st August in each year, in order that the List may be made as correct as possible.]

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CASE

IN WHICH THE

VENA CAVA INFERIOR

WAS OBSTRUCTED FROM THE COMMENCEMENT OF THE COMMON ILIAC VEINS, AND ITS CAVITY ENTIRELY OBLITERATED BETWEEN THE ENTRANCE OF THE EMULGENT AND HEPATIC VEINS.

By THOMAS BEVILL PEACOCK, M.D.,

PHYSICIAN TO THE ROYAL GENERAL DISPENSARY, ALDERSGATE STREET.

Communicated by ALEXANDER NASMYTH, Esq.

Received October 28th—Read November 12th, 1844.

Though a considerable number of instances are recorded, in which the main venous trunks of the upper or lower portions of the body have been found more or less completely obstructed, it is conceived that the following case, which furnishes an unusual example of an entire obliteration of the inferior cava, will be interesting to the Society.

Agnes M'Ewen, aged 47, of intemperate habits, was admitted into the Royal Infirmary of Edinburgh, on the 16th of August 1842; her friends stated that she had been married at the age of 25; had had six children, and always recovered well after her confinements. In the October preceding her admission into the Infirmary, her health began to decline, and the catamenia appeared more frequently, and the discharge was more copious than previously. In the

course of the spring she received a severe blow on the abdomen, and the following day had hæmorrhage from the bowels. After this her health rapidly gave way, and the catamenia ceased; the abdomen became swollen, and one month before her admission her legs also were observed to be œdematous, the urine being at the same time scanty and high-coloured. Two days before her death she was seized with profuse vomiting of blood, and when received into the Infirmary was greatly prostrated, her countenance was pale, puffy, and sallow, the limbs very œdematous, and the abdomen large and tense. Shortly after being removed to the ward, the hæmatemesis recurred, and bloody fluid was discharged from the bowels. She sank eight hours after her admission.

The autopsy took place thirty-seven hours after death.

The body was very anasarcous, and the abdominal cavity contained ten pints of a pale straw-coloured fluid.

The right lung was adherent to the costal and diaphragmatic pleuræ by old cellular attachments. The left lung was universally adherent, and at the base of the lung the false membrane had undergone the fibro-cartilaginous degeneration: it was full half an inch in thickness, and of a glistening white colour; the lung was of small size, and the corresponding portion of the chest was contracted. Both lungs were œdematous. The heart was natural, weighing $9\frac{3}{4}$oz. avoirdupois: its muscular substance was pale. The epithelium of the mucous membrane

of the œsophagus, near its termination, allowed of being readily removed.

The stomach contained a large quantity of a thin bloody fluid, but scarcely any coagula; the mucous membrane when washed was of a pale yellowish white colour, except in one or two places where there existed slight redness, which as the membrane was throughout of natural firmness, allowing of being drawn off in streaks of the usual length, was regarded as resulting from imbibition: no ulceration or abrasion was anywhere detected. The small and large intestines were healthy, the glands of Peyer were slightly elevated above the adjacent mucous membrane, and distinctly defined.

The liver was extremely small, weighing only 24oz.; but, with the exception of some opacity of its serous coat, was free from disease; numerous large venous branches ramified on its convex surface.

The spleen was large, weighing 9½oz., and moderately firm; several small extravasations of blood were found in its substance.

The left kidney was reduced to a very small size, weighing only 1½oz.: it was scabrous and lobulated, of a pale clay colour, and covered with numerous small cysts; the pelvis was expanded, and the striated portion almost entirely destroyed. The right kidney was large, weighing 5½oz.: it was of a pale colour, and on section the granular deposit was found extensively infiltrated into the striated portion, and between the pyramidal bodies.

The uterus was somewhat larger than usual, and

was attached by firm adhesions to the rectum, sigmoid flexure of the colon, and the sides of the pelvic cavity.

On removing this organ from the body, the veins in its substance, in the broad ligaments, and around the ovaries, were found distended by firm fibrinous clots, and on following the venous branches, in the course of the circulation, the common iliac veins displayed similar coagula, and they thence extended continuously throughout the cava, till, about 1½in. below its passage through the diaphragm, that vessel became reduced to an impervious ligamentous cord. The fibrinous clots on the right side, though firmly obstructing the common iliac vein, ceased immediately above its division; but on the left side they extended both into the hypogastric and external iliac veins, entirely closing their cavities. The left common iliac vein was firmly adherent to its corresponding artery.

In the veins of the uterus and ovaries, the coagula had a peculiar fusiform shape, and were firmly adherent to the coats of the vessels, generally by one extremity: they were very solid, so large as to distend the vessels, and generally about half an inch in length, and were composed of lymph in layers of a pale colour, alternating with others of a pinkish hue. In the iliac vessels and cava they consisted of continuous masses, distinctly laminated, not unlike the fibrinous layers of an aneurismal sac. Their colour in the iliacs and lower part of the cava was pale red, and they gradually became more completely decolorized

as they advanced towards the heart; they were closely united to the tunics of the veins, and entirely filled their cavities. The spermatic veins were of large size, and their coats thicker and firmer than usual; the orifice of the left vein into the trunk of the emulgent, and of the right into the cava, were closed by firm fibrinous masses, and the course of the right spermatic vein was occupied by small fusiform clots adherent to its coats, and similar to those of the uterine veins.

The calibre of the iliac veins, and especially of the cava, was much reduced, and their coats were indurated and thickened: this became more conspicuous towards the upper part of the vessel;—the cavity occupied by the clots gradually diminishing, and the coats increasing in thickness, till, as before stated, about an inch and a half below the passage of the vessel through the diaphragm, it became entirely obliterated, and was reduced to a mere cord, about the size of the little finger, of a glistening white colour, and firm fibrous texture.

The communication of the left emulgent vein with the cava was open, and this vessel was throughout free from coagula; a vein of considerable size, also unobstructed, entered it from above. The right emulgent vein was closed by a large and closely adherent coagulum, projecting into its orifice.

The heart, with the liver, and other abdominal viscera, having been removed from the body before the obstruction of the cava was detected, I was unable to institute so satisfactory an examination as

would have been desirable, into the course by which the venous blood of the lower extremities had been returned to the heart. On examination, however, the vena azygos was found of unusually large size, being immediately above the entrance of the vena azygos minor, fully one inch in circumference; the branches entering the right vein, and the vena azygos minor with its branches, and especially some from the spinal canal and intercostal spaces, were greatly enlarged. The portal trunk and branches were free from disease; the hepatic veins entered the only portion of the vena cava inferior which remained pervious.

Though the convex surface of the liver was covered by enlarged veins, the diaphragmatic veins were not materially dilated.

The veins of the integuments of the abdomen, and those following the course of the internal epigastric artery, did not appear larger than usual. Those of the upper extremities and head were free from any appearance of disease. But little blood was contained in the cavities of the heart, or in the vessels. It was everywhere of a very pale colour, and almost entirely fluid.

The above case possesses several points of interest:—

1. It evinces the facility with which the venous circulation can be maintained notwithstanding the entire obstruction of one of its main trunks. In this respect it is not less remarkable than the case related by Dr. Baillie, in which "the inferior cava

was changed into a ligamentous substance, from the entrance of the emulgent veins to the right auricle of the heart," yet we are assured that this condition was in no degree accessory to the death of the patient. Cases of this description, in which the obliteration of the vessels is complete and of long duration, are decidedly rare, though not a few instances are recorded of partial or recent obstructions in the cavities of the inferior and also of the superior cava. The history of the present case is unfortunately very defective: the swelling of the abdomen, we are informed, was not observed till three or four months before the death of the patient, and the legs became œdematous only one month before that event; these symptoms may therefore fairly be ascribed to the atrophied condition of the liver, and advanced state of disorganization of the kidneys, rather than to the disease of the veins, since the entire obstruction of the upper part of the cava, and its conversion into a firm, white, fibrous cord, must, it is conceived, have required for its completion, a much longer period. The formation of the obstruction in the veins was most probably due to an attack of uterine disease, of which we possess no history, and which probably occurred some time anterior to the symptoms which preceded the fatal event. The absence of any report of such affection will excite little surprise in those familiar with the defective and erroneous statements of hospital patients and their friends.

2. There could be little doubt that the course

of the venous circulation had been entirely re-established before death, though the exact means by which the blood from the lower extremities was returned to the heart could not be fully traced, from the advanced stage of the examination when the obstruction of the cava was detected. The observations which were made, render it, however, probable that the largest portion found its way into the superior cava through the vena azygos. That vessel was found greatly dilated, being fully double its ordinary size, and the intercostal branches, and those proceeding from the spinal canal, were also much enlarged. The freedom of anastomosis between both divisions of the vena azygos, and the spinal and lumbar veins, would afford a ready channel for the returning column of blood, which may have reached those vessels through the medium of the ileo-lumbar and circumflexa ilii veins. The communication between the left emulgent vein and the commencing branch of the vena azygos minor would allow the return of the blood from that vessel.

Though the branches of the vena porta freely anastomose with the systemic veins, more especially through the medium of the vena mesenterica inferior, and may thus, in cases when the cava is obstructed below the entrance of the hepatic veins, assist in maintaining the circulation, it is not probable that in the present instance this channel was materially concerned in conveying the blood from the lower extremities to the heart. The liver, it will be seen, though free from other morbid change, was very

much reduced in size, being less than half its usual weight; the portal circulation must therefore have been much impeded, and hence, most probably, the dilatation of the venous branches on the surface of the liver. Mr. Kiernan* has shown that the capsular veins of that organ are branches of the portal system, and anastomose with the phrenic veins, and thus become the medium of a collateral circulation, in some cases of atrophy of the liver. M. Reynaud,† in a case of obstruction of the vena cava and portal trunk, found the veins on the surface of the liver dilated, and traced them to a common trunk which pierced the diaphragm, and opened into the cava above the seat of the obstruction.

There seems no reason to suppose that the integumental veins of the abdomen and thorax, and the epigastric, thoracic, and mammary veins, materially contributed to form collateral channels for the conveyance of the blood.

3. The larger or smaller venous trunks may become obstructed either as the result of inflammation of the vascular tunics; by pressure exercised on their coats by aneurismal or other tumours; by the accumulation of morbid matters, fungoid or cancerous, in their cavities; or, lastly, by the coagulation of the contained blood from retarded circulation through diseased organs, or in peculiar states of system. Instances of obliterations of the cava traceable to

* The Anatomy and Physiology of the Liver. Philosophical Transactions, 1833, p. 733.

† Journal Hebdomadaire de Médecine, tom. v. p. 173.

these several causes are referred to in the Appendix. In the case narrated in the paper, the disease was evidently inflammatory in its origin, though from the defective history of the patient's previous state of health it is not easy to assign the period at which the inflammation had occurred. From the same cause it is impossible to ascertain the relation which the disease of the veins bore to the affection of the kidneys, which was the immediate cause of death. The frequency of affections of the endocardium lining the cavities and covering the valves of the heart, and of the inner coat of the aorta, in persons who have died from the effects of granular degeneration of the kidneys, has been dwelt upon by all writers on that disease:—the similar tendency to secondary affections of the venous system appears however to have attracted little attention. In 1835 Dr. Osborne* described the emulgent veins as occasionally found obstructed by coagula in cases of granular degeneration, and the same observation has since been repeated by Rayer,† and Stokes,‡ and more recently by Dr. Chevers,‖ who also refers to the not unfrequent occurrence of phlebitis of the veins of the extremities in persons labouring under renal disease. I have three or four times found firm, partially decolorized, and more or less adhe-

* The Nature and Treatment of Dropsical Diseases, 12mo. Dublin, 1835.

† Maladies des Reins, tom. ii. p. 104. Paris, 1840.

‡ Dublin Journal, vol. xxi. p. 144. 1842.

‖ Guy's Hospital Reports, vol. viii. p. 95.

rent coagula, in the veins of diseased kidneys; and have twice observed phlebitis of the femoral and inguinal veins occurring during the course of the chronic form of disease, and after death have found those veins obliterated by fibrinous exudations adherent to their parietes. It seems, therefore, that phlebitis is by no means an uncommon complication of renal disease;* and it may be inferred that the lining membrane of the vascular system generally, like the serous membranes to which it bears so close an analogy, is peculiarly liable to inflammatory action in persons labouring under that affection.

4. The examination of the mucous membrane of the stomach and intestines afforded no explanation of the source of the hæmorrhage which occasioned the death of the patient;—no ulceration, abrasion, or even congestion being anywhere detected. The absence of the signs of disease of the mucous membranes where intestinal affections have been predominant during life, appears to be not unfrequent in the bodies of persons who have died of disease of the kidneys. I have found the mucous membrane pale, of natural firmness, and free from any appearance of disease, throughout the whole canal, in patients who were mainly carried off by the secondary diarrhœa and dysentery.

* Dr. Bright, in the first volume of his Medical Reports, has related the case of a young woman who died of renal dropsy, and in whom the portal vein and its hepatic branches, with the splenic vein and the pulmonary artery, were filled with firm clots, partially deprived of red particles. Case 6, p. 19.

APPENDIX,

CONTAINING REFERENCES TO THE CASES OF OBSTRUCTION OF THE SUPERIOR AND INFERIOR CAVA, PREVIOUSLY PUBLISHED.

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1. *Obstruction resulting from inflammation of the coats of the veins.*

INFERIOR CAVA.

By far the larger number of cases of obstruction of the inferior cava are of this description, and specimens exhibiting the disease are contained in several of the Museums in London. In all the preparations, however, which I have had an opportunity of examining, and probably in nearly all the published cases, the change was recent, and the cavity of the vein was obstructed by fibrinous masses. The case recorded by Dr. Baillie, and that described in the paper, afford, so far as I am aware, the only examples in which the trunk of the inferior cava was converted into an impervious cord. Probably in most cases, as in that related, the disease has commenced in the uterine veins, and been thence extended into the cava.

Baillie,—Transactions of a Society for Improvement in Medical and Chirurgical Knowledge, vol. i. p. 127.—The vein converted into a ligamentous cord, from the entrance of the emulgent to the hepatic veins. Collateral circulation assisted by two veins following the course of the vena azygos.

Wilson, in ibid. vol. iii. page 65.—Recent obstruction of the vein the sequel of puerperal phlebitis.

Haller,—Pathological Observations, London 1756, p. 41, and Philosophical Transactions, vol. xliv. 1746 & 47, p. 527.—Vein closed by a fibrinous mass from the emulgent veins to its division into the iliacs. Collateral circulation by the right spermatic vein.

Reynaud,—Journal Hebdomadaire de Médecine, tom. v. p. 173.—Obstruction extending for three inches below the auricle, and involving also the hepatic veins and the right branch of the vena porta. The coats of the veins thickened, and fibrinous deposit on their exterior. Collateral circulation through the external abdominal veins.

Journal Universel et Hebdomadaire, tom. ii. p. 381.—Fibrinous clots extending from the entrance of the emulgent veins to the iliac and femoral veins, but not producing complete obstruction. The vena porta contracted and containing coagula.

Laennec,—Archives Générales de Médecine, tom. vi. p. 619.—A preparation of the inferior cava and right iliac vein obstructed by clots exhibited to the Académie de Médecine de Paris, at the desire of M. Laennec.

Knape,—Hufelands Journal der Practischen Arzneikunde und Wundarzneikunde, vol. xxxvi. p. 12.—The vein closed by the adhesion of its coats.

Gross,—Pathological Anatomy, Philadelphia, vol. i. p. 295, notice in Dublin Journal of Medical Science.—Vein closed by a plug of lymph from the

second lumbar vertebra to the liver. Clots in the portal vein, and some of the veins of the left lung.

Bright,—Medical Reports, vol. ii. p. 66, Case xxiv.—Coagulum, yellow and laminated, and in some places presenting an appearance of suppuration, occupying the cava and iliacs from beneath the liver to Poupart's ligament.

Carswell,—Fasciculus of Plates illustrating the formation of Analogous Tissues, plate 2nd, fig. 5.—Lower portions of the cava and the iliac veins obstructed by adherent fibrinous masses.

Rayer,—Maladies des Reins, plate 50, fig. 4.—The cava and emulgent veins obstructed by clots, the sequel of puerperal phlebitis.

SUPERIOR CAVA.

Complete obstructions of the superior vena cava are extremely rare : of the nine cases to which I am able to refer, three only appear to have been complete. The most frequent cause of obstruction in this vessel is the pressure of aneurismal or other tumours. The following cases may probably be the result of inflammation :—

Lobstein,—Anatomie Pathologique, t. ii. p. 110.—The vein closed by a solid mass, which by its pressure had caused obstruction of the two right pulmonary veins,—quoted from Weissbrod's Observat. Anat. Duæ.

J. Reid,—Edinburgh Medical and Surgical Journal, vol. xliii. p. 297.—The vessel converted into a fibrous cord. Cause of obstruction not ascertained,

but it is suggested to have been possibly owing to the pressure of diseased bronchial glands.

Breschet,—French Translation of Hodgson on Diseases of the Arteries, &c., t. ii. p. 480, note.—A model contained in the Musée de la Faculté de Médecine de Paris, of an obstruction of the cavity of the superior cava by a fibrinous mass, prepared under the direction of M. Dupuytren.

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2. *Obstructions resulting from pressure on the trunks of the veins.*

INFERIOR CAVA.

Cline,—Wishart's Trans. of Scarpa on Aneurism, note, p. 20.—The vein obliterated shortly above its bifurcation, by a tumour originating behind the peritoneum.

Bouillaud,—Archives Générales de Médecine, tom. vi. p. 567.—The lower portion of the vessel obstructed by the pressure of diseased mesenteric and lumbar glands.

Andral,—Clinique Médicale, Diseases of the Liver.—Partial obstruction of the inferior cava and porta by the compression of diseased lymphatic glands.

SUPERIOR CAVA.

Reynaud,—Journal Hebdomadaire de Médecine, tom. ii. p. 110.—Imperfect obstruction from the pressure of an aneurism.

Dr. W. Hunter,—Medical Observations and Enquiries, vol. i. p. 333.—Similar case.

Also Otto,—Neue Seltene Beobachtungen, Berlin 1824, part i. p. 65,

And Deckart, in Otto's Pathological Anatomy, by South.

Dr. Wilson, in Report of Proceedings of London Med. and Chir. Soc., London Med. Gazette, vol. xxi. p. 666 (vol. i. for 1837 & 38).—Two cases of imperfect obstruction of the superior cava from pressure: one of these, in which the compression was produced by an aneurism, is more fully described by Mr. Shaw, Med. Gaz., vol. xxvi. p. 522 (vol. ii. for 1839 & 40). Dr. Watson alludes to a case of this kind in his lectures, probably that of Dr. Wilson. I have seen the left vena innominata and the trunk of the vena azygos entirely obliterated from the pressure of aneurisms, and the right iliac and inguinal veins filled with fibrinous clots from the compression of the vein by an uterine tumour.

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3. *Accumulation of morbid deposits in the cavity of the veins.*

CAVA INFERIOR.

Cruveilhier,—Anatomie Pathologique, Livraison 5, p. 3 & 4.—Medullary matter filling the vena cava as far as the kidney, in a person from whom the testis had been removed;—a band of fungoid glands found along the lumbar portion of the spine.

Rayer,—Maladies des Reins, plates 47 & 49.—Vena cava inferior and emulgent vein, filled with pale

deposit in a case of fungoid disease of the kidney; the deposit in the veins was probably of a similar nature to the disease of the kidneys.

Barlow,—Guy's Hospital Reports, October 1844.—Vena cava inferior distended with medullary matter, and its lower extremity contracted in connection with fungoid disease of the kidney. Fungoid matter is probably only found in the veins, when the same disease affects adjacent organs, and chiefly in the smaller veins of the organs so diseased. Cruveilhier, in Livraison xviii. plate 1, has figured an obliteration of the hepatic vein by medullary deposition; and Carswell has given drawings of the same in the portal and renal veins, and in the veins of the stomach. I have seen the trunk of the vena porta and its branches in the liver filled with a pale deposit, probably carcinomatous, in a case of medullary sarcoma of the liver.

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4. *Obstruction of the veins by coagula resulting from stasis of the contained blood.*

This cause of venous obstruction is scarcely likely to operate in the main venous trunks. The only examples to which I can refer are the clots found in the veins of the liver and kidneys, in some forms of chronic disease of those organs in which the coats of the veins present no appearances of inflammation. The obstructions in the veins of the extremities occurring in persons exhausted by long continued diseased action, or great loss of blood, may probably be

in part ascribed to the enfeebled circulation combined with a peculiar condition of the blood. In most of these cases, however, a certain degree of excited action in the affected vessel appears to attend the formation of the coagula; though the number of vessels consentaneously affected show how much this excited action is connected with general causes. See cases detailed by Dr. Bright in his Medical Reports, vols. i. & ii.; and also the cases of Reynaud and Gross.

Lobstein has described a case in which he found the veins of a limb long paralysed, contracted, and containing clots, which he ascribed to the coagulation of the blood from loss of nervous energy. He says, " Cette derniére cause m'a paru évidente dans un cas d'hemiplégie qui s'est terminé par la mort. Les parois du veines du côté paralytique étaient toutes plus ou moins obstruées par des polypes, tandis que celles du côté sain renfermaient du sang dissous et fluide."—Traité d'Anatomie Pathologique, tom. ii. p. 610.

In the Compendium of Pathological Anatomy by Otto, there are references to one or two cases which I have not been able to find: he also refers to the following:—

Rhodius,—Mantissa Anatomica, obs. xxi. p. 15.—The trunk of the vena cava so completely obstructed that it would not allow the passage of a probe: " haud dubie ab exulceratione a vermibus in ramo iliaco repertis excitata."

Bartholinus (Th),—Hist. Anat. Rar., cent. ii. hist. 35.—The vena cava obstructed near the heart.

Bontius,—De Medicinâ Indorum, fol. 37.—The vena cava closed by a fatty substance.

Albinus,—Annotationes Academicæ, lib. 7, c. 9. —The vena cava obliterated near the origin of the iliacs.

In a note to Portal's Anatomie Médicale, t. iii. p. 437, Chopart is stated to have seen in England an obliteration of the inferior vena cava, in which the circulation was maintained by means of the epigastric and internal mammary veins.

A fuller account of several of the cases here referred to is given by Dr. W. Thomson, in vol. xliii. of Edinburgh Medical and Surgical Journal. See also Rokitansky, Handbuch der Pathologischen Anatomie, ii Band. p. 674.

ON THE

CLASSIFICATION, STRUCTURE AND DEVELOPMENT

OF THE

ECHINOCOCCUS HOMINIS,

SHOWING REASONS FOR REGARDING IT AS A SPECIES OF CYSTICERCUS.

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Received November 4th—Read November 12th, 1844.

Observations on the echinococcus hominis have been heretofore so rare, that I deem myself fortunate in having the opportunity of communicating to the Society some original researches relative to the habits, structure, and development of this curious entozoon.

My observations, hitherto, have been limited to two cases of hydatids, instances of the common acephalo-cyst of the liver, in both of which I discovered the animalcule in question. I was led by this circumstance to infer that the echinococcus must be more common in its occurrence than had previously been imagined, and I find my opinion corroborated by that of Mr. Curling. In a note recently received from this gentleman, he says, "As the echinococcus has been discovered in nearly all the cases of acephalo-cyst which have come under my observation during

the last two or three years, I am inclined to think that this, instead of being, as I once supposed, a rare form of hydatid, is the most common one infesting man."

Dr. Livois, the author of a work, lately published in Paris, entitled, "Recherches sur les Echinococques,"—(a work which I regret that I have been unable to obtain, but of which a notice appeared in the British and Foreign Medical Review for January 1844,)—considers them to exist in every case of acephalo-cyst.

From these statements it is obvious that the echinococcus is a common parasite of the human frame, and that it is probably present in the majority of instances in which acephalo-cysts occur. On this account, therefore, independently of the scientific interest connected with the knowledge of a curious animal, I have concluded that a description of the habits, structure and development of this parasite would be deemed acceptable by the Society; and to this subject I at once address myself.

The term echinococcus (εχινος, echinus, κοκκος, granum) was applied by Rudolphi to the acephalocyst and its contained animalcules in conjunction, and in this sense the term has been used by Mr. Owen in his article "Entozoa" in the Cyclopedia of Anatomy and Physiology, and by Mr. Curling in the Society's Transactions. Müller* employs the term in its more significant meaning, as referrible to the animalcules

* Archiv fur Physiologie, &c. 1836. Jahresbericht im Jahre 1835, page 107.

alone, and Mr. Owen in a later work* adopts the same application of the term. The question is not unimportant, for the former use of the term assumes a speciality in the acephalo-cyst, while the latter recognises the animalcules as independent beings or as parasites of the common acephalo-cyst. My own observations lead me to the latter conclusion, and they further go to prove that the echinococcus has no claim to consideration as a distinct genus, but is in reality a species of the genus cysticercus, to which species, for reasons hereafter to be detailed, I think the term "*cysticercus pedunculatus*" peculiarly applicable.

The acephalo-cyst in which the echinococcus is found, is identical with that in which the animalcules are absent. Nothing is more true than the fact stated by Müller and admitted by Dr. Livois, that in some acephalo-cysts the animalcules are present, while in others, from the same subject, they are absent, and there may be cases in which they are wanting in all the cysts, although such cases are probably rare. The acephalo-cyst is composed, as is well known, of two tunics—an outer tunic, which is semi-transparent, laminated, finely granular in minute texture and highly elastic; and an internal layer, (fig. 1,) consisting of a thin and very delicate membrane studded with innumerable transparent cells, varying in extremes of measurement from

* Lectures on the Comparative Anatomy and Physiology of the Invertebrate Animals, 1843.

$\frac{1}{10000}$ to $\frac{1}{300}$ of an inch in diameter; but having a medium size of $\frac{1}{2000}$ of an inch.

This membrane is the seat of development of the echinococcus, and to this, in a fresh acephalo-cyst, they are found connected by means of a very delicate proper membrane, either singly or more commonly in clusters, varying in numbers of individuals from two to one hundred (fig. 3). Some idea of the numbers of echinococci inhabiting a single acephalo-cyst of moderate size, may be inferred from the fact, that in one cyst of the size of a small hazel-nut I counted forty clusters; in several of the clusters there were eighty individuals, and in the entire cyst not less than one thousand. Now, when it is recollected that the majority of acephalo-cysts in an ordinary hydatid tumour would be larger than the one here described, and that of these there may be from one hundred to one thousand present, the entire number of living beings nourished at the expense of the fluids of the diseased person, in such a case, must be enormous.

Acephalo-cysts are generally very closely packed in an hydatid tumour, so that they cannot be removed without some degree of pressure; from their roundness of form and distension they are not easily brought under examination; and when punctured, their fluid contents issue from the sac in a jet of considerable force, impelled by the contractile power of their elastic tunic. Moreover, if the incision be of a certain size, the sac will roll up and turn itself inside out. These are all reasons why the little

clusters connected to the internal membrane of the acephalo-cyst should be found detached in the fluid of the cyst, and broken up into their individual components; a state which I conceive to be the result of violence, but which has been regarded by all authors who have written upon them, as their natural condition when their development is completed. Müller, in referring to the adhesion of the clusters to the internal surface of the acephalo-cyst, remarks, that he conceives this contact to have relation to development.

The echinococcus presents itself to the observer in two states—a contracted and an elongated state. In the former of these, (figs. 4—8,) which, according to my observations, (limited to the dead animal,) is the most common, the echinococcus is more or less globular or oval in form, and slightly flattened at opposite poles. In the latter, namely, the elongated condition, (figs. 9—11,) it offers considerable variety of shape, being usually larger at the cephalic than at the caudal end, displaying generally an apparatus of suctorial prominences and hooklets at the cephalic extremity, being more or less constricted in the middle, and frequently pointed at the caudal end. The ordinary dimensions of the animal in its contracted state are $\frac{1}{200}$ of an inch in the long, and $\frac{1}{230}$ of an inch in the short, diameter; while the largest specimen which I have yet seen in the elongated state measured $\frac{1}{125}$ of an inch in the long, and $\frac{1}{190}$ in the broadest part of its short diameter.

In structure, the animal is a mere integument, the one half, representing analogically the head and neck, being susceptible of retraction into the other half, which represents the body or caudal portion; the proper contents of the tegumentary sac being a small number of nucleated granules, and an apparatus (of the existence of which I am not certain) for the retraction of the head and neck. The *head* is a flat membranous disk which forms the extremity of the neck, and has imbedded in its substance an apparatus of small hooks disposed in a circle. Immediately behind the head are four rounded prominences, of considerable size, placed at equal distances from each other, and consisting each of a prominent rim surrounding a depressed cup; these are *suctorial processes*. Beyond the suctorial apparatus follows the body or *caudal portion* of the animal, and at the extremity of the latter is a short process, the *peduncle*, (figs. 4, 7,) by which the animal is attached to its own proper membrane or to the internal membrane of the acephalo-cyst.

The integument of the animal is white, thick and opaque (during life, according to Dr. Livois, it is perfectly transparent); it is also pliant, contractile, moderately tough, and composed of nucleated granules. It is thicker in the neck than in the body, and the suctorial prominences are transparent and *structureless*. When the animal in its contracted state is viewed under the microscope by transmitted light, a dark line may be seen occupying the centre of its longitudinal axis and extending for a variable

distance from one pole towards the other; this line is the tubular canal of the inverted neck. At the extremity of this line is another, which is transverse in its direction and curved; this latter line indicates the position of the circle of hooklets, and presents some variety in accordance with its direction. The appearances which I am now describing are more distinct in some specimens than in others, they are also better seen when the animal is subjected to moderate compression or to the action of diluted acetic acid. When rendered transparent by the latter means, the entire neck, with its suctorial prominences, may often be distinguished within the body. In this view of the animal also, when the head is only partially retracted, I have sometimes seen running from the base of the head to the posterior pole of the body, some delicate lines which appeared to me to be the boundaries of a retractor muscle of the head. This impression is strengthened by observing that in complete contraction of the echinococcus, the posterior pole of the body is drawn in and rendered concave; the entire animal having then the shape of an orange.

When the animal in the contracted state is viewed in its horizontal axis, (fig. 6,) the circle of hooklets appears as a ring in the centre of the body; an outer and inner circumferential line denote the thickness of the integument, and not unfrequently the suctorial prominences may be distinguished as transparent spaces. In this position, if the animal be transparent, the circle of hooklets may be

brought into focus in such a manner as to give the appearance of being on the surface of the body.

The elongated state of the animal may be ascertained, if not by its length, at least by its irregularity of form. Viewed in its longitudinal axis, the coronet of hooklets, and two or three suctorial prominences, may be perceived, while in its horizontal or oblique axis, more or less of the four suctorial prominences may be distinguished, with the coronet of hooklets in their midst.

The hooklets are thirty-four in number, seventeen long, measuring $\frac{1}{1050}$ of an inch, and seventeen somewhat shorter (fig. 17). Each hooklet, (figs. 18, 19, 20,) possesses a gentle curve, is pointed at one end and blunt at the other, and presents a convex and a concave border, and a base which encroaches on the concave border for nearly half its length: the base is moreover distinguished in this aspect by a breadth greater than the rest of the hook, and by a bifid process at its central extremity. This bifid prominence in the base of the hooklet is a considerable and distinct process in the larger forms of cysticercus, in which also the base has the character of an elongated shaft, and the hook is more strongly curved.

I have said that the neck of the animal is terminated by a membranous disk, which is susceptible of assuming the three states of convexity, concavity, or simple flatness. In this membrane the hooklets are imbedded, a small portion only of the point of each projecting free beyond the integument. When the

disk is viewed in its flat state, the hooklets are seen to be placed on the same plane, their blunt extremities being directed towards the centre of the disk, and their shafts radiating towards its circumference (fig. 17). In this position of the disk, the base of the hooklet is directed towards the body of the animal, and the bifid process at its central extremity is deeply and firmly inserted into the substance of the neck, the point of attachment of this part constituting a kind of pivot upon which the hooklet moves. The movements of the hooklet are such as serve to place it in an erect or depressed position; in the former, the blunt end is drawn backwards, while in the latter it is pushed forwards, in both cases the bifid process being the fixed point upon which the motion is accomplished. The more common position of the hooklets is that in which they are depressed or recurved; in this position the blunt ends are directed forwards, while the sharp ends look backwards, and the convex border outwards (figs. 9, 11). The erect position of the hooklets is less frequent; in this the direction of the ends is reversed, and the concavity of the hook with its base, and the bifid process of the latter, look outwards (fig. 10).

I now come to that character of the echinococcus which I consider worthy of becoming a specific distinction, namely, the *peduncle*, by which the animal is fixed, either to its own proper membrane, or to the internal membrane of the acephalo-cyst. The peduncle is cylindrical, short and granular. When

the animal is separated from its attachment, the peduncle offers a variety of appearances, depending on the manner in which it may be torn, and when it is broken close off, an opening is left, which communicates with the internal cavity of the body of the animal. Through this opening I have frequently seen nucleated cells expelled by compression (fig. 8 g). The peduncle has been noticed by Müller, but is regarded by him as a stage in the development of the animal needing further elucidation.

In the course of pursuing the observations on which the preceding account of the echinococcus is founded, I frequently met with animals that had been dead apparently for some time. Some of these retained their elongated form (fig. 11), and were more or less wrinkled and shrivelled; others were contracted, but had lost their characteristic orange or pumpkin-shaped form, and exhibited a number of transparent cells, apparently adipose cells of various magnitude, scattered irregularly through their substance (figs. 12, 13); others again were reduced to a mere mass of cells (figs. 14, 15), and could only be identified by the remains of the circle of hooklets being apparent in some; while others, still further advanced in degradation and much smaller, had the appearance of the corolla of a flower with a single circle of petals (fig. 16). Occasionally I met with instances in which an entire cluster still contained in their proper membrane had undergone these changes, but not all of them in the same degree, so that I was enabled to observe all the grada-

tions here described, within the circuit of the same membrane. Indeed, without such a confirmation as this observation afforded me, I should have been unable to speak positively to the identity of bo d is so very unlike their original form.

Development.—In my description of the internal membrane of the acephalo-cyst, I have stated that this membrane is studded with innumerable transparent cells, but amongst these cells I have observed here and there one which was more opaque than the rest. All the cells, both transparent and opaque, contain smaller cells, but, in the latter, the contained cells are more defined and generally nucleated. Such a cell as that which I am now describing, measured $\frac{1}{2000}$ of an inch in diameter (figs. 21, 22); another measured $\frac{1}{900}$ of an inch (fig. 24); while between these there was every transition of size. These compound nucleated cells I conclude to be the earliest stage in the development of the echinococcus.

The second stage of development appears to result from the rupture of the membrane of the preceding cell, and the diffusion of the contained nucleated cells in the form of a small opaque patch in the substance of the internal membrane of the acephalo-cyst. Two patches of this kind measured, the one $\frac{1}{600}$ (fig. 25), the other $\frac{1}{480}$ of an inch in breadth, and were composed of nucleated cells measuring $\frac{1}{3000}$ of an inch.

The next change which occurs is the protrusion of the superficial stratum of the lining membrane of the acephalo-cyst by multiplication of the cells of

the central part of the patch. In this stage the little mass of cells forms a globular prominence on the surface of the membrane, and one cell of large dimensions may be distinguished by its transparency in the centre of the rest; the membrane which invests the little mass is the enclosing membrane of the future animal. A small globular mass, such as I am now describing, measured $\frac{1}{500}$ of an inch in diameter (fig. 26). Another of greater length, which assumed a pyriform shape, measured $\frac{1}{275}$ of an inch in its long diameter (fig. 27); another, more decidedly pyriform, $\frac{1}{250}$ of an inch; and a third $\frac{1}{175}$.

While this process is going on, as respects the first developed animal, the patch from which it originated is increasing in size, and other protrusions are taking place around its base, which go through the same phases and produce other individuals (fig. 28). From this circumstance, animals in various stages of development may be seen in the same cluster, and enclosed in the same proper membrane.

With elongation of the embryo, the large central cell also enlarges, and the cells in the central axis of the embryo all assume a larger size than the rest. Subsequently these larger cells appear to coalesce, and then a tubular canal occupies the central axis of the embryo, commencing by an enlarged expansion in the bulbous extremity of the animal, and terminating by a similar expansion at its point of connection with the opaque patch (fig. 29). At the distal end of the tubular canal a dark transverse body appears by degrees: this is the future circle of hook-

lets ; and from it, some lines, indicating the situation of the neck, may be traced to the extremity of the animal, and the aperture for the eversion of the neck begins to be formed (fig. 30).

I regard as not the least interesting in relation to the development of this animal, the question,—What becomes of the tubular canal which is primitively formed in the interior of the embryo ? I believe the answer to this question to be, that this tubular canal remains as a natural part of the organization of the perfect animal ; that in the latter it is modified so as to perform the office of a retractor muscle ; and that a portion of this same tubular cord, extending beyond the periphery of the animal, maintains the permanent character of peduncle. This view of the nature of the peduncle will explain the formation of the aperture in the posterior pole of the animal when the peduncle is torn off, and the consequent escape of granules through the opening when the body is compressed.

It may be expected that I should say something in explanation of the functions of the organs possessed by the animal ; but concerning these I can offer no satisfactory hypothesis. The creature has no mouth, and is therefore nourished by imbibition ; it is pedunculate, and has no organs of progression. But what purpose can the hooklets fulfil ? I can only reply to this question by asking,—What purpose do they fulfil in the cell-bound cysticercus cellulosus, or in the gigantic cysticerci of quadrupeds ? What can be the use of suctorial organs to a pedunculate

animal? The only solution of which these questions admit, is, that the type of organization which nature bestows on a particular group of animals is uniform, however different the circumstances of individual animals may be;—that the mammary glands, for example, are a type of a large class of animals, but perform an office only in a particular sex.

On the identity of the echinococcus with the cysticercus, it is unnecessary to dwell. The characters of the genus are, a head provided with hooklets, a neck supporting four suctorial processes, and susceptible of retraction within the body, and a membranous cyst-like body. All these characters are present in the echinococcus: the situation, the number, the general form of the hooklets, and their arrangement, are the same in both, and the suctorial processes are the same. The neck, it is true, is shorter in echinococcus than in cysticercus, and the cyst-like body thicker and less expanded; but these are merely comparative differences. The peduncle however is an important distinction, and merits, I think, the notice bestowed on it in this paper. In describing the structure of the animal, I referred to the probable existence of a retractor muscle. Nothing like a retractor muscle has, as far as I know, been described in the cysticercus; but in a cysticercus which I examined recently, I found such a muscle, and have prepared it for preservation.

*Figures illustrating the structure and development of the Echinococcus hominis.**

Fig. 1.—A portion of the internal membrane of an acephalo-cyst, magnified 155 times. The irregularities on its surface are cells varying in diameter from $\frac{1}{10000}$ to $\frac{1}{300}$ of an inch, the common size being $\frac{1}{2000}$.

Fig. 2.—A group of cells remarkable for their circular form, and for the large size of the included secondary cells. They measured $\frac{1}{1250}$ of an inch in diameter, and were associated with numerous small globular cells, measuring $\frac{1}{6000}$ of an inch. The figure is magnified 310 times.

Fig. 3.—Groups of echinococci, contained within a delicately thin proper membrane; by which they are connected with the internal membrane of the acephalo-cyst. Magnified 38 times.

Fig. 4.—A small portion of the proper membrane of the echinococci, showing the pedunculate connection between these animals and the membrane The point of attachment is the posterior pole of th animal.

Fig. 5.—An echinococcus, as seen in its longitudinal axis, the head retracted; magnified 155 times. *a*. The infundibiliform aperture of the tubular canal, formed by the retraction of the head. *h*. The circle of hooklets and head. *r*. The retractor apparatus of the head. *o*. The caudal aperture caused by the

* See Plate I.

rupture of the peduncle. *s. s.* Two of the suctorial processes seen through the semi-transparent integument of the animal. *t.* The granular tegument.

Fig. 6.— An echinococcus viewed transversely; the head being directed towards the observer. In this figure the membranous disk, which the hooklets encircle, is seen. *s. s.* Suctorial processes. Magnified 155 times.

Fig. 7.—An outline figure, showing the animal with the head partly retracted. *p.* A portion of the peduncle.

Fig. 8.—The animal more retracted, having three of the suctorial processes apparent through the transparent parietes. *g.* Some of the granular contents of the animal, expelled through the aperture left by the torn peduncle, and still remaining connected with it.

Fig. 9.—An animal showing the head entirely extended; the four suctorial processes seen. *d.* The membranous disk encircled by the hooklets, rendered convex by the contraction of the neck.

Fig. 10.—An animal in a similar state, exhibiting an erect condition of the hooklets.

Fig. 11.—Two specimens of the echinococcus, shrivelled and deformed.

Figs. 12-16, Exhibit states of degradation of the animal, occurring after death. The form of the creature is lost; large transparent cells (adipose cells?) are seen in its interior, and finally it is reduced to a small cluster of nucleated particles, re-

cognisable only as the remains of the animal by the presence of the circle of hooklets; as in fig. 14.

All the figures from fig. 5 to fig. 16 are magnified 155 times.

Fig. 17.—The circle of hooklets seen upon its under surface, and magnified 456 times. The hooklets are thirty-four in number; seventeen long, and seventeen short.

Fig. 18.—Lateral views of the separate hooklets. *b.* The base. *c.* The central extremity, or bifid process of the base.

Fig. 19.—Hooklets viewed upon the concave or inferior border. *c.* The bifid process of the central extremity of the base.

Fig. 20.—A diagram, illustrating the movements and position of the hooklets. The dotted line represents the outer surface of the neck, and runs through the fixed point of the three hooks. *f.* A hooklet in the erect position, as in fig. 10. *g.* A hooklet in the horizontal position, as in fig. 17. *k.* A hooklet in the depressed or recurved position.

Figs. 21-30, Illustrate the development of the echinococcus; figures 21-24 are magnified 210 times; and figures 25-30, 155 times.

Figs. 21-25.—Masses of nucleated granules; in the interior of figure 23 a large nucleated cell is seen.

Figs. 26, 27.—The nucleated masses assuming a pyriform shape. They are connected by a disk of smaller granules to the membrane, from which they

grow, and exhibit a transparent cellular space in the centre.

Fig. 28.—A cluster of several pyriform masses of granules; in each the transparent centre is perceptible.

Figs. 29, 30, Exhibit a more advanced stage of development; in which the head is formed, and the canal for the extension of the head is seen.

Fig. 31.—A sketch of four echinococci; two perfectly formed, and two in progress of development, connected by peduncles with the same granular spot.

A CASE

OF

ANEURISM OF THE POPLITEAL ARTERY,

CURED BY COMPRESSION OF THE FEMORAL ARTERY.

By EDWARD GREATREX, Esq., Surgeon,

AND

W. T. C. ROBINSON, Esq.,

ASSISTANT SURGEON OF THE COLDSTREAM GUARDS.

Communicated by EDWARD STANLEY, F.R.S.,

PRESIDENT OF THE SOCIETY.

Received December 2, 1844—Read January 14th, 1845.

On 2nd May 1844, Private John Hedley, ætat. 27, 1st Battalion, Coldstream Guards, walked to the Hospital and complained of pain and swelling behind the right knee, which had obliged him to fall out of the ranks on returning from a quick drill in Hyde Park.

On examination, a large irregularly-shaped aneurism was found filling up the popliteal space, strongly pulsating, and admitting of being partially emptied by pressure. It was tender and painful, the leg and foot somewhat swollen, and veins turgid. The swelling prevented his straightening the knee-joint, the circumference of which, taken over the middle of the patella, was 17¾ inches, but of the sound knee only 15½ inches. He had perceived the

tumour only a few days before his admission into the Hospital.

His health has been always good since he enlisted four years ago, as well as before that time, when he was a farm servant: his height 6ft. 1½in., weight 14st. 4lb. Pulse 84, natural, and, excepting the aneurism, he has every appearance of strong health, and is reported to be a man of regular habits.

Rest in bed, and some opening medicine, relieved in a few days the pain in the tumour, and the slight œdema of the foot.

When he had been a week in the Hospital he was attacked by acute laryngitis, which required active treatment, blood-letting, &c., and in a few days after recovery from this alarming seizure, a repetition of similar measures was necessary, together with blisters and mercury, until ptyalism occurred, to subdue a sharp pleuritic inflammation of the right side.

On his convalescence in the beginning of June, it was intended to treat the aneurism, which had meanwhile slowly enlarged, in the ordinary manner by ligature of the superficial femoral artery; but by Mr. Guthrie's recommendation, the treatment lately so successful in Dublin was adopted, and simple pressure applied on the vessel.

The prevalence of erysipelas in the Hospital was an additional inducement to prefer this advice, to the operation.

An instrument was made for the purpose by Mr. Weiss. It consisted of an Italian tourniquet, a broad short splint to fit the outer and back part of

the thigh being substituted for one of the usual pads, as less likely to cause pain and sloughing.

It was first applied on the 18th of June, on the artery at about six inches below the fold of the groin, and the pulsation of the aneurism stopped; but on visiting him shortly afterwards it was found to beat again with unabated force, and this was the case over and over again, although the shape of the pad was changed, and various other little alterations tried.

He had now the ill luck to be attacked by modified small pox, which, although slight, occasioned necessarily some relaxation in the treatment of the aneurism.

The same constant escape of the vessel from beneath the pad occurred until the 8th of July, notwithstanding pressure was made as strongly as he could bear it, or as was advisable for fear of sloughing. The knee then measured $18\frac{3}{4}$ inches, and the tumour had become softer on the outer side, the pulsation and bellows-sound being very strong and loud. On the 8th of July the plan was adopted of screwing the pad down firmly, and leaving him the key, so that he might, when the pain became intolerable, and "burning like a hot iron," to use his own words, relax the pressure by turning the screw for an instant or two, taking care always to apply his thumb or fingers with all his force on the artery just above, in order that during these short but frequent intervals the passage of the blood should still be retarded, if not stopped.

On the 9th of July this method was found to have been successful, the tumour was now perfectly solid, and no pulsation, or bellows-sound has since been detected in it.

The instrument was kept applied as described, for nine days after the pulsation had ceased, to avoid any risk of the stream of blood again making its way into the aneurism: on the afternoon of the 18th of July it was entirely removed, Mr. Stanley agreeing in the opinion that all danger had passed. The skin under the pad was slightly inflamed, and three or four small blisters had risen there; beyond this, there was no damage done by the instrument. The superficial femoral artery was clearly felt to pulsate down to its entrance into the tendinous canal, though perhaps not quite so forcibly as in the opposite thigh. Two arteries as large as crow-quills employed in the collateral circulation, were felt passing downwards on the surface of the now hard and solid tumour.

On the 9th of August, the tumour having diminished, he was allowed to walk about on crutches. On the 24th he walked well with a stick, and could put his heel to the ground. From this time the progress of the case may be told in a few words. He has been kept in the Hospital, but allowed to be up and about all day. Absorption of the contents of the aneurism has proceeded very slowly; with a view of hastening it, friction with stimulating liniments, and gentle pressure by the application of a flannel bandage, have been used.

On the 8th November the measurements were, of the right knee 16¾ inches, of the other 15½ inches; although he can perfectly straighten the joint, he finds it easier to walk with it slightly bent. The tumour in the ham is hard, flattened, and yet nearly as large as a hen's egg: on its surface the vessels above mentioned are still perceptible. His general health has been very good, and he has grown fat and stout. On the 14th November he was dismissed the Hospital, and returned to his battalion in the Tower, to undertake light duty.

January 18th, 1845.—He performed the light duty until the 12th December, when he was placed on full duty, which he has efficiently discharged ever since. The tumour does not now feel as it did after pulsation had ceased, namely, dull and solid, but rather elastic, and has undergone a further diminution, the circumference of the knee being about a quarter of an inch less than it was when he was discharged the Hospital. He has continued in good health, and is an active man.

P.S.—The following was the state of the patient on the 6th of June, as reported by Mr. Robinson in a letter to Mr. Greatrex:—"I measured Hedley's knee this morning—it is 15¾ inches around—the other 15 inches,—precisely the same dimensions as when you last saw the man. The tumour to the touch is much the same as when you left town: it gives him no inconvenience. He is stout and well, and does all his duty."

ON

EXTRAVASATIONS OF BLOOD

INTO THE

CAVITY OF THE ARACHNOID,

AND ON THE FORMATION OF THE FALSE MEMBRANE WHICH SOMETIMES ENVELOPS THESE EXTRAVASATIONS.

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The extravasations of blood treated of in this paper have of late years been carefully examined by several French pathologists, as one of the forms of their "Apoplexie des Méninges;" but these affections have not, as far as I am aware, received from English pathologists that degree of attention which they will, I think, be found to deserve.* As I have had opportunities of investigating several of these extravasations, and the various appearances arising from them, many of which are closely linked to some of the most important points in Pathology, I trust that the following observations will be acceptable to the Society.

Blood extravasated into the cavity of the arach-

* At the time when this was written, I was not aware that Dr. G. Burrows had, in his Croonian Lectures, published some cases and observations which are very analogous to some of the observations mentioned in this paper.—See Dr. Burrows' Croonian Lectures, 1835, Medical Gazette, vol. xvi. p. 710.

noid membrane may there be found in different states, which, for the sake of perspicuity, I shall arrange in four divisions, according to their degrees of simplicity.

1. The extravasated blood may be either liquid or coagulated; if in the latter state, it may be in clots, or spread out in the shape of a thin membranous layer, covering a greater or lesser extent of the surface of the brain.

2. Sometimes the extravasation presents itself under the shape of a false membrane, possessing more or less of the original colour of the blood; in some cases it is even reduced to the fibrine only, and of a slightly yellowish tinge.

3. The blood may be fixed to the free surface of the arachnoid, and there maintained by a membrane, which to the naked eye presents all the characters of the serous membrane itself.

4. The blood is frequently found enclosed in a complete cyst, of various degrees of thickness, which may be removed unbroken from the cavity of the serous membrane.

The four divisions above referred to may be, and often are, combined with each other, but, in whatever state the extravasated blood has been found, it has, in the majority of cases, corresponded to the upper surface of the brain, and has been rarely met with in the cerebellic fossæ.

FIRST DIVISION.

The main feature of this division is, that it forms

a most important link in the appearances about to be examined; for in these cases there never has been any difficulty about defining the exact nature and situation of the disease.

Case 1.—J. W., ætat. 45, was admitted into St. George's Hospital, in the beginning of 1841, under Dr. Hope, in an advanced stage of phthisis, of which he died, a few days after his admission. During the residence of this patient in the Hospital, he was repeatedly attacked with delirium.

Examination 39 *hours after death.*—Covering the free surface of the parietal arachnoid, which lines the right middle fossa of the skull, was a large coagulum of blood, measuring, in some places, two lines in thickness. This coagulum, of a fawn colour towards its cerebral surface, was flattened and spread out, but it had not contracted any adhesions with either of the layers of the arachnoid. The brain and its membranes did not present any other trace of disease; the source of the hæmorrhage was not discovered. No marks of external violence existed in any part of the skull.

Cases of this kind may be found in various authors. In one of the *three* cases so often quoted from Rostan,* as cases "of extravasation of blood between the parietal arachnoid and the dura mater," it is distinctly stated by that author, that the blood was effused into the *cavity* of the *arachnoid*, where it was found in large dark-coloured clots. Abercrombie†

* Rostan, sur le Ramollissement du Cerveau. Obs. 91.

† On Diseases of the Brain, p. 243.

mentions a case of this nature in which the extravasated blood was closely adherent to the inner surface of the dura mater, from which it could be peeled off like a membrane; the brain was healthy.

SECOND DIVISION.

The thin, membrane-like layer of coagulated blood, mentioned in the preceding paragraph, evidently gives rise to the appearances noticed in this division. Once thus spread out, the coagulum after some little time loses its colouring matter; the fibrine, of different hues, is left, and then gives rise to a false membrane, of various degrees of thickness.

The membranous appearance which coagula sometimes put on, in the cavity of the arachnoid, has been known to pathologists for many years. Among Sir B. Brodie's preparations, there is one, put up in 1826, which is thus described:*—"Layer of coagulated blood, presenting the appearance of a false membrane on the under surface of the dura mater."

The following case affords a good illustration of the appearances which these extravasations of blood sometimes give rise to.

Case 2.—S. S., ætat. 73, was admitted into St. George's Hospital under Mr. Tatum, on the 23rd of July 1844, with concussion of the brain, and a small lacerated wound over the external angular process of the right frontal bone, which however

* Catalogue of Sir B. Brodie's Museum, D. 2.

was not exposed. There was some bleeding at the nose, and the eye-lids were very much ecchymosed. At the back of the right hand, there was a large lacerated wound, exposing the extensor tendons. Shortly before her admission she had been run over by a cab.

She soon recovered from the effects of the concussion, and then vomited some blood which, it appeared, had been swallowed. For some days she was confined to her bed by sloughing of the wound on the hand, and inflammation of the absorbents, accompanied by low, typhoid symptoms, but she rallied, and was ultimately able to go about the ward. She was about to be discharged from the Hospital, when erysipelas appeared on the hand, which soon began to slough again. Typhoid symptoms came on, and she fell into a state of torpor, from which she was, however, easily roused, and answered all questions very well; there never was any paralysis. In this state she continued for some days, and died on the 20th of September.

Examination twenty-nine hours after death.—In the cavity of the arachnoid, on either side of the falx, were the remains of a very extensive effusion of blood. On the left side, the remains of the extravasation consisted of a firm, membranous layer, which lined the parietal arachnoid, upon which it was gradually lost, corresponding to the whole of the upper surface of this hemisphere of the brain. This false membrane, of a reddish brown colour, and a line in thickness, was divisible into thin layers, between which, in some places, there were some

circumscribed cysts, containing bloody serum and coagula. On the right side, the extravasation occupied a much greater extent than on the left; it corresponded to the lateral parts, and to the middle fossa of the skull, as well as to the whole of the upper surface of the hemisphere. At the lateral and inferior parts, the extravasated blood formed a large, firm, dark coagulum, two lines in thickness, enveloped in a firm membrane of a yellowish colour, but at the upper part it presented appearances similar to those observed on the left side, with the exception of the membrane being thicker, and having in its centre one large cyst only. These membranes were united, in the whole of their extent, by one of their surfaces, to the parietal arachnoid; on the other surface they were perfectly smooth and polished, presenting to the naked eye all the characters of a true serous tissue, but they were easily detached, with the cysts and coagula in their structure, from the serous membrane. These membranes were throughout their whole structure most plentifully supplied with blood-vessels. The visceral arachnoid presented its usual polished appearance, and it was neither thickened nor discoloured. The brain was healthy, both in colour and in structure; the main arteries at its base presented several patches of atheromatous deposit. The source of the hæmorrhage was not discovered. There were no marks of violence about the external parts of the skull.

The false membranes, arising from extravasation

of blood, may be found loose in the cavity of the arachnoid, attached to one of the free surfaces of that membrane; or they may become a bond of union between its two layers, and this, too, without the intervention of any inflammatory action.

In the great majority of cases, however, the false membrane is connected with the parietal arachnoid only, to which it is pretty firmly attached; its free surface, corresponding to the visceral arachnoid, being perfectly smooth and polished, and presenting all the characters of a serous tissue; under these circumstances it is generally supplied plenteously with blood-vessels. When thick, this membrane is oftentimes divisible into distinct layers; of which, the external ones are of a light colour, whilst the internal ones still possess more or less of the colour of the blood. Clots of blood, of various sizes and colours, are not unfrequently found, either in the structure of the membrane, or upon one of its surfaces. These clots may have proceeded either from the vessels of the arachnoid, or from those of the adventitious tissue. Cysts containing serum and blood, or serum alone, are also met with in these membranes.

The newly-formed tissue is at times soft and pulpy; at other times having existed for a long period, and undergone various changes, it presents a fibrous, a leathery, and even a cartilaginous appearance. In these states, if firmly attached to the parietal arachnoid, the membrane is very likely to be mistaken for diseases of a very different character;

thus, the appearances now under consideration have been referred to chronic inflammation, either of the dura mater, or of the arachnoid, with thickening of these membranes; they have also been described as the result of inflammation of the meninges, producing an exudation of coagulable lymph upon the free surface of the arachnoid.

In Mr. Cæsar Hawkins's Museum, there is a preparation* which will, I think, serve to illustrate this point very well. It consists of a large portion of the dura mater from an adult, with extensive false membranes, attached to the parietal arachnoid, which are supposed to have been the result of inflammation ending in an exudation of lymph; but, from a careful examination of the preparation, I am of opinion that these membranes are the result of extravasations of blood, of long standing. On the right side of the falx, the false membrane is composed of three distinct layers, the two external ones being pellucid and of a white colour, the central one being about two lines in thickness, opaque, and of a mottled colour, yellow in some parts, and of a brownish red in others. On the left side of the falx, the false membrane has not been separated into layers; it is about two lines in thickness, opaque, and white, except in patches where it is of an orange colour. The free surfaces of these membranes are for the greater part smooth, but in some places they present a flocculent appearance.

I am inclined to think that Dr. Abercrombie's

* Catalogue of Mr. Cæsar Hawkins's Museum, J. a. 5.

Case* "of remarkable thickening of the dura mater, with separation of its laminæ, and a deposition of a new matter betwixt them, with a cavity containing yellowish serous fluid," belonged originally to an extravasation of blood into the cavity of the arachnoid, rather than to a chronic inflammation of the dura mater. The history of the case, the symptoms, and the morbid appearances, belong precisely to these extravasations; especially when they have been, as this was, of long standing. At the time at which that case was drawn up, but little attention had been paid to the accurate definition of the affections of the various membranes of the brain. The false membrane being closely connected with the parietal arachnoid, and having, from the time of the existence of the disease, (two years,) undergone various changes, it may, I think, be fairly supposed, that Dr. Abercrombie was misled as to the true seat of the morbid deposit.

Dr. Bayle† has minutely described, under the head of "chronic meningitis with false membranes in the cavity of the arachnoid," 13 cases, in some of which, if not in all, the membrane owed, I think, its origin to an extravasation of blood, and not to an exudation of lymph the result of inflammation, as that author supposes. To this opinion I have been led by a careful perusal of the descriptions given by Dr. Bayle, as well as by other considerations. In every one of Dr. Bayle's cases, the false membrane was either of a red colour, or it presented

* Diseases of the Brain, p. 49.

† Maladies du Cerveau et de ses Membranes.

the various hues which are known to depend upon the colouring matter of the blood; in the majority of the cases, clots of blood, of various sizes, were mixed with the false membrane; in some of them it is distinctly stated that the membrane presented the appearance of a thin and flat coagulum, whilst in others the description tallies exactly with the appearances which will be hereafter described in the Third Division. To account for the frequent admixture of blood, which Dr. Bayle found in these cases, he has been driven to the supposition, that the nature of the inflammatory action had changed at different periods, so as at one time to produce an albuminous secretion, then a secretion of blood, followed by another albuminous secretion; now it is by no means so common an occurrence, as these cases would lead us to suppose, to find *clots of blood* mixed with the effusions resulting from inflammation.

Another circumstance which must be taken into consideration, is the comparative rarity of inflammatory effusions into the cavity of the arachnoid, whereas extravasations of blood have often been met with in this situation, since the attention of pathologists has been directed to this affection. Mr. Baillarger,* who has had frequent opportunities of examining these membranes, under circumstances precisely similar to those of Dr. Bayle, has also come to the conclusion that, in the majority of these cases, the membranes are the result of an extravasation of blood, of more or less standing.

* Bulletins de la Société Anatomique de Paris, 1834, p. 86.

THIRD DIVISION.

In this division have been classed the cases in which the extravasated blood is fixed to the free surface of the parietal arachnoid by a fine, delicate, transparent membrane, apparently possessing all the characters of the serous membrane itself, of which it, at first sight, appears to be a part. Of these appearances, the two following cases are good examples.

Case 3.—In the early part of 1841, Mrs.——— ætat. 65, after having for several days suffered from great mental excitement, was suddenly seized with violent pain confined to the right brow, which lasted for two or three hours, and then disappeared. This pain continued for several days to recur twice in the twenty-four hours, presenting all the characters of brow ague; it was apparently relieved by quinine, but it was followed by a train of low symptoms, accompanied by a dry brown tongue, wandering and impairment of the intellectual faculties, terminating in coma. There never was any paralysis, neither were any contractions of the limbs observed. The patient died twelve days after the first attack of pain.

The body was examined about twenty-four hours after death. The veins of the scalp were gorged with blood. The dura mater, on the right side, appeared to be somewhat thicker than natural. A large quantity of blood was found extravasated in the cavity of the arachnoid; it corresponded both to the upper and to the inferior surfaces of the right hemi-

sphere, and a small quantity of it had even made its way into the corresponding cerebellic fossa, where it was lying on the margin of the foramen magnum; the whole of this extravasated blood was of a dark fawn colour. At the base of the skull the extravasation at first sight appeared to have taken place between the dura mater and its arachnoid lining, for the blood was covered by a thin, smooth and polished membrane, presenting to the naked eye all the characters of the serous tissue, with which it was perfectly continous, at the margins of the extravasation; but this membrane and the clot were easily removed, and the arachnoid was then found slightly roughened, but uninterrupted throughout in its continuity. The substance of the brain was healthy, but its ventricles contained some fluid. The source of the hæmorrhage was not discovered. No marks of external violence existed about the cranium.

Case 4.—J. G., ætat. 25, was admitted into St. George's Hospital, in July 1842, under Dr. Wilson, in an advanced stage of phthisis, of which she died a month after her admission. During her residence in the Hospital, this patient had frequent attacks of violent delirium; her father had died maniacal, and for some time previous to her death she had been addicted to drinking.

The body was examined eighteen hours after death. The dura mater was somewhat more firmly adherent to the calvaria than usual. In the cavity of the arachnoid, and corresponding on either side of the falx to the centre of the upper surfaces of

both hemispheres of the brain, was a thin layer of coagulated blood firmly attached to the parietal arachnoid, where it was confined by a thin, delicate membrane of a fawn colour, presenting towards its free surface all the characters of the original serous tissue, to which it was most perfectly adapted; but these appearances were ascertained to be produced by a false membrane similar to that described in the preceding case; and after the removal of the coagula, the arachnoid was found slightly roughened and discoloured. The cells of the pia mater were filled by an extensive effusion of serum; the substance of the brain was healthy. The source of the hæmorrhage was not discovered: no marks of external violence existed about the cranium.

This class of cases derives an increased degree of interest from the fact that these extravasations of blood were for many years described as having taken place between the dura mater and the parietal arachnoid, which membranes were thus supposed to have been widely separated from each other. Cases of this nature thus described, may be found in the works of Rostan, Andral, Cruveilhier, Dr. Hodgkin, and several other authors.

Dr. Hodgkin,* in 1836, having alluded to Rostan's cases as a strong proof of the existence of a parietal arachnoid, gives the details of a case of this nature, which had occurred at Guy's Hospital, upon which

* Lectures on the Morbid Anatomy of Serous Membranes, p. 83.

he makes the following observations: "The extravasated blood presented, for the most part, a smooth, polished surface towards the visceral arachnoid, and was covered by a thin, even, firm and generally transparent membrane, which, in some parts, *seemed to be double*, having the effused blood between its layers. This delicate membrane could be nothing else than a portion of arachnoid reflected over the dura mater, and giving it its polished surface. Though very thin, it was too firm and resisting, to be regarded as a false membrane formed from the fibrine of the effused blood. The dura mater, when this membrane was peeled off, lost its natural polish, and exhibited its fibrous texture."

It is with diffidence that I venture to offer an opinion contrary to that expressed by Dr. Hodgkin; but, since the publication of the case above referred to, the result of the investigations which have been carried on by several pathologists and by myself, is so directly opposed to this view, that I cannot but think that Dr. Hodgkin has been led into an error, of which the case itself will, I think, be found to bear internal evidence; for, supposing this membrane to have been a portion of the parietal arachnoid stripped off from the dura mater, I cannot account for that *membrane* appearing to be in some parts *double*.

The pathological investigations carried on within the last few years by Messrs. Longet, Baillarger, Calmeil, Ernest Boudet, and others, have all shown that the fine, delicate membrane which covers these extravasations of blood, and which presents to the

naked eye all the characters of a serous tissue, is a newly-formed membrane, so beautifully adapted to the original serous membrane, that it is only with the utmost care that the exact limits of each can be defined. The cases of this nature which I have examined, have afforded me an opportunity of verifying the accuracy of this opinion; and in speaking of the formation of this membrane, I shall bring forward several examples, which will, I trust, prove that it is much more frequently and much more rapidly formed than is usually supposed.

Blood extravasated, and thus connected to the parietal arachnoid, may present itself under two different aspects: it may either be collected in one cavity; or it may be disseminated in patches, of various sizes and thickness, over the surface of the serous membrane, the intervening parts of which present a natural aspect: of the first kind, cases three and four are good illustrations; of the latter kind I have lately examined a very good specimen, in an injury of the brain, with extravasation of blood into the cavity of the arachnoid—the patient lived nine days after the accident, and each patch of blood was found already covered by its own membrane. In Guy's Museum there is a beautiful specimen, described in the catalogue "as patches of blood effused between the dura mater and its arachnoid lining," which, I think, belongs to the class of cases at present under consideration. To this kind, I am also inclined to refer the patches, of various sizes, of a reddish brown or dark yellow colour, described by

Dr. Foville* as separating the arachnoid from the dura mater; with which patches, this author in another paragraph states that extravasations of blood are not unfrequently connected. The patches are, I presume, nothing but these disseminated extravasations of blood, which have lost more or less of their colouring matter. Even Dr. Foville, although he classes these appearances under his "Méningite pariétale," states that their inflammatory nature is not sufficiently well established, for him to enter into a detailed account of the various circumstances connected with them.

In this class of cases the collections of blood have never, that I am aware of, been found fixed to the free surface of the visceral arachnoid; even where the blood has proceeded from a laceration of the brain, I have found it fixed to the parietal layer of the serous membrane.

FOURTH DIVISION.

The true nature and situation of those collections of blood, described by many pathologists as being situated between the dura mater and its arachnoid lining, are most easily detected in the cases classed in this division, where the blood is contained in a completely closed bag, which may be detached from the parietal arachnoid, and, with its contents, removed unbroken from its situation.

The existence of these encysted collections has

* Dict. de Méd. et de Chir. Prat.—Article, "Méningite."

been known to pathologists for many years,* but their identity with the supposed extravasations of blood between the dura mater and the arachnoid was, as far as I know, first pointed out by Mr. Baillarger in 1834. The appearances which these collections of blood present, are well exemplified in the following case.

Case 5.—E. W., ætat. 51, was admitted, on the 10th of March 1844, into St George's Hospital, under the care of Dr. Wilson. At the time of the admission of this patient, he was nearly in a state of insensibility, with some low muttering delirium. Both the inferior extremities were partially paralysed, the right more so than the left: the right superior extremity was also similarly affected—the hand of this side was blue and swollen. The pulse was remarkably slow and weak: the tongue white: the bowels open: there was no distention of the bladder. It was stated by his daughter, who brought him to the Hospital, that, for the last two years, he had been complaining of his head, but that he had always been able to continue his occupation, as a carrier, up to within the last six days, when he was taken ill on the road, as he was coming with his waggon from Cromer, from which period he had been confined to his bed. It was further stated that, of late, he had had a good deal of anxiety. After having been in the Hospital for a few days, he rallied so far, as to be able to answer all

* Catalogue of Sir B. Brodie's Museum, 1826, D. 16.

the questions which were put to him; but his intellectual faculties still remained weak, and his memory was very defective. He also regained some power over his inferior extremities, which he was able to draw up in bed. During the period of his existence in the Hospital, a marked alternation was observed in the symptoms, which, some days, appeared to be greatly relieved, and at other times much increased, with affection of the speech, and paralysis of the bladder. Towards the latter part of his life, the symptoms put on a typhoid character, in which state they remained until the death of the patient, which took place on the 16th of April. The day before his death, he so far rallied as to be able to recognise his daughter. The treatment consisted of local bleeding, with purgatives, and gentle stimulants. The preceding notes were taken from Dr. Wilson's case-book.

On the examination nineteen hours after death, I found the frontal bone studded, in several places, with deposits of a scrofulous nature, which had, apparently, been originally situated in the diploë, from whence they had, in some parts, caused the absorption of both tables, and in other parts that of the external table only. The external surface of the dura mater presented no morbid appearances worth noticing. Connected with the parietal arachnoid were two remarkable collections of blood, one on either side of the falx. At first sight, these extravasations appeared to have taken place between the dura mater and its serous lining, dissecting off the

latter to a great extent; but, after a careful dissection, it was ascertained that these appearances depended upon a false membrane, which formed over each extravasation a complete cyst, connected by one of its surfaces to the parietal arachnoid; whilst the other surface, being perfectly smooth and polished, presented all the characters of the original serous tissue, to which it was so beautifully adapted, that it was with difficulty ascertained where the respective membranes began. The connections of the left cyst with the serous membrane were somewhat looser than those of the right, so that that cyst was more readily peeled off from the arachnoid, which was found somewhat roughened, but not discoloured. Each of the false membranes extended far beyond the cysts, and lined the greater part of the parietal arachnoid, corresponding to the upper surfaces of the hemispheres; but the right cyst was much larger, and much more prominent, than the left. The walls of the cysts were much thicker than any other part of the false membranes, which were bevelled off, and gradually lost upon the serous tissue; on the left side, the cyst was about a line in thickness. The right membrane was of a yellowish brown colour; the left was of a light yellow. The cavity of the right cyst contained bloody fluid and coagulated blood, amounting in all to about ʒiv; the coagula presented various colours—some were dark, some of a rusty, and others of a yellow-ochre colour; the cavity of the left cyst was not more than a quarter of the size of that of the right,

and its coagula were much more solid. Both cysts were perfectly smooth on their internal surfaces, except at a few points, where some fibrinous coagula were adhering to them. The margins of the membrane on the right side presented, at their junction to the serous tissue, a network of vessels, most beautifully injected, which proceeded in countless numbers towards the cyst. The visceral arachnoid was throughout perfectly sound, and unconnected with either cyst; but on the right side, both the brain and its investing membranes were deeply tinged with yellow. The hemisphere of the brain, corresponding to the right cyst, presented a deep cup-like surface. The substance of the brain, with the exception of the discoloration above mentioned, was healthy in its aspect, but its ventricles were distended with serum. The superior longitudinal sinus, and the upper part of both lateral sinuses, were perfectly healthy. The source of the hæmorrhage remained undiscovered. There were no marks of external violence about the skull.

Encysted collections of blood, such as those described in the preceding case, have been found in various parts of the cavity of the arachnoid; but in the great majority of cases, these collections correspond to the upper surface of the hemispheres of the brain. The connections of these bags with the free surfaces of the serous membrane in which they are lodged, are sometimes so slender that they appear all but loose; at other times, these bags are pretty firmly connected, both with the parietal and with

the visceral arachnoid, but most frequently they are connected with the parietal arachnoid only, to which they are sometimes most beautifully adapted; and, in this case, so much do they, on their free surface, partake of the character of an original serous tissue, that they appear, at first sight, to be a part of diseased and thickened arachnoid stripped off the fibrous membrane.

If the disease be of some standing, the cavity of the cyst may be perfectly smooth and polished, or it may be intersected by fibro-cellular bands, running in various directions: sometimes fibrinous clots are found adhering to this internal surface.

After a certain period, the membranes forming these cysts become thoroughly supplied with blood-vessels, which may be seen in countless numbers, permeating their whole structure; in several cases it has been noticed, that a minute network of vessels, most beautifully injected, is found at the margins of the new tissue, thus marking the point of union between the original and the false membranes. Thus organized, these cysts possess all the physiological characters of an original serous membrane; they secrete, they absorb; they have been found filled with clots of fibrine and blood-tinged serum; sometimes they contain serum alone, of various colours, and oftentimes, in the cavity of the same cyst, are found coagula of blood of various hues,* some recently effused, and others of long standing. In one case, given by Abercrom-

* Case No. 5; and Ernest Boudet, "De l'hémorrhagie des Méninges," p. 23.

bie,* all the contents of the cyst had disappeared, and the cyst itself was found flattened and perfectly collapsed. Some French pathologists think that the cavities of these cysts may be obliterated by adhesions taking place between their walls; but with such appearances as those described, I see no possibility of deciding whether a large cyst ever did exist, or whether the false membrane did not originally belong to the cases already alluded to in the Second Division of this paper.

I cannot dismiss this part of the subject, without alluding to the great similarity which exists between these cases and those described by Dr. Foville, as the result of true arachnoiditis. So little do the accurate descriptions, given by Dr. Foville, resemble those of the inflammatory effusions observed by other pathologists, and so much do they resemble the cysts formed round extravasations of blood into the cavity of the arachnoid, that I cannot but think that they must have belonged to this class of cases. In each case the cyst was of a *yellow* colour; in five cases out of the six, it contained serum only, but in the sixth it was filled by a *considerable quantity of blood.*

In some cases, where encysted collections of blood have been found in the cavity of the arachnoid, it has been noticed that the visceral arachnoid, pia mater and brain have been blood-stained. Mr. Calmeil† thinks that this discoloration may be made use of, as a means of deciding whether the

* Loc. cit. p. 277. † Dict. de Méd., 2e édit., p. 462.

cyst preceded or followed the extravasated blood. I do not think that this test can be admitted as a valid one; for, if blood extravasated into the cavity of the arachnoid can stain the pia mater and brain, why should it not produce the same effect by transuding through the walls of the cyst? what is there to prevent transudation from taking place through a false membrane, as easily as through an original one? This transudation, it is well known, often takes place after death: of this I observed a good example some short time back, in which an extensive extravasation of blood into a complete and thick cyst, situated below the fascia of the neck, did not, during life, produce any discoloration of the skin; but in a few hours after death, the skin, in the neighbourhood, became extensively discoloured.

In the history of these encysted extravasations in the cavity of the arachnoid, one of the most interesting studies is that of the formation of the false membrane, so often alluded to, by which the extravasated blood is subsequently, either partially or wholly, surrounded, as in the Third and Fourth Divisions of this paper.

The most generally-received opinion is, that an exudation of lymph, consequent upon the arachnoid being irritated by the presence of the blood, takes place, and surrounds the extravasation; this explanation may serve for some few cases, but it will certainly not do so for the majority. I am convinced that the opinion, broached by some pathologists, of the membrane being, *in some cases*, formed from the

blood itself, will be applicable to *a very large majority of these extravasations*; and I shall now bring forward several examples which, I trust, will tend to prove that this membrane is wholly formed by the coagulated fibrine of the extravasated blood, and not by a secretion of lymph, as is usually supposed. To avoid any misconception on this point, I have carefully selected cases in which there were no appearances of inflammation either during life, or after death.

Blood extravasated into various serous cavities, and covered over by a false membrane, without the least trace of inflammation in the neighbouring tissues.

In a patient, who lived nine days after an extensive laceration of the brain and its investing membranes, I found, in the cavity of the arachnoid, on the left side, a large quantity of recently-effused blood, part of which was fluid; the greater part, however, was coagulated, and divided into a visceral and a parietal portion. The visceral portion formed a firm, smooth, and flattened clot, of about two lines in thickness, covering the whole of the arachnoid of the upper surface of the cerebral hemisphere, with which it was merely in contact. The parietal portion was formed by clots, of various size and thickness, firmly attached to the free surface of the parietal arachnoid by a thin, delicate membrane, of a fawn colour, which had a smooth and polished surface, and, at its margins, was bevelled off, so that

it appeared to be continuous with the original serous tissue, part of which it might have been taken for, on the supposition that the blood had been effused between the dura mater and its serous lining. The clots and their coverings were, however, easily removed with the nail, and the arachnoid was then found but slightly roughened by the contact of the blood.

In the left cerebral ventricle of a woman who died with symptoms of apoplexy, I found a large, solid coagulum of blood, enveloped in a fine, transparent cyst, perfectly polished on its free surface, except at the part where it was adherent to the membrane covering the corpus striatum, and anterior part of the thalamus: these adhesions were easily destroyed with the finger. In the structure of the membrane forming the cyst, was a most delicate net-work of vessels, beautifully injected; the blood contained in the cyst was quite solid, and of a variegated appearance, being in some parts of a fawn colour, in some of a yellow ochre, and in others of a deep red brown. The other parts of the ventricle were filled with a recent effusion of blood, the source of which was traced to the substance of the thalamus. The membrane lining the ventricle was slightly thickened, and of a deep yellow ochre.

In 1841 I examined the body of a man who died a few days after having been tapped for dropsy, depending on cirrhosis of the liver. The serum which was in the cavity of the peritoneum was deeply tinged with blood. Coagula of blood were

in various parts of the abdomen: several of these were situated on the anterior abdominal parietes, in the neighbourhood of the wound made by the trocar in the linea alba. These coagula, of a dark fawn colour, were covered over, and maintained in their situation by a fine transparent membrane, perfectly continuous with the parietal peritoneum, and presenting all the characters of a true serous tissue. At first, it appeared as if the blood were situated between the fascia transversalis and the peritoneum, but this appearance was easily destroyed by the removal of the coagula and their false membranes, and the peritoneum was then found only slightly roughened by the contact of the blood. There were no traces of inflammation about the peritoneum.

In July 1844, a man was admitted into St. George's Hospital, under Mr. Cutler, for a severe injury of the chest, with laceration of the lung, after which he lived five days. In the cavities of both pleuræ were large quantities of bloody fluid; on the pleuræ costales were several large clots of dark-coloured blood fixed to this situation by a transparent whitish membrane, perfectly smooth on its free surface, and presenting the appearance of the pleura having been extensively stripped off, with the blood situated behind it; but the clots and their membranes were easily removed, and the pleura was found still firmly adhering to the subjacent tissues. In some points the false membrane formed a complete cyst, containing the thinner part of the blood, which was easily removed unbroken from its situa-

tion. There were no signs whatsoever of inflammation in either pleural bag.

A middle-aged man was admitted into St. George's Hospital, under Mr. Cutler, in October 1844, with a severe injury of the chest. He lived eleven days after the accident, six of which he was in the Hospital, and during this time he never presented a single inflammatory symptom. The cavity of the left pleura, completely filled with bloody fluid, of a deep colour, was subdivided into two compartments by a portion of coloured fibrine, presenting a honeycombed appearance, which passed from the ribs to the lung; the lower compartment was itself subdivided into several lodges, by other layers of coloured fibrine intersecting each other. Large portions of loosely-coagulated blood were found in all these cavities; some of these clots were of a rusty colour, others approached nearer to the natural colour of the blood. The lung was compressed against the spine, and the whole surface of the pleural sac was coated by a false membrane, about two lines in thickness, formed by coagulated fibrine. The fibrine which lined the pleura pulmonalis was, towards its free surface, loosely coagulated, but towards the lung it was dense, firm, and of a yellowish colour: that which lined the greater part of the pleura pulmonalis and pleura diaphragmatica presented, on its inner surface, a smooth and polished appearance, and in colour exactly resembled the yellowish fibrine found in the clots of the heart of this patient. So uniform was this coating,

and so continuous was it, throughout its whole extent, that it looked, at first, merely like thickened pleura; but this appearance was easily destroyed by peeling off this adventitious membrane from the serous tissue, which then presented the same appearances as the pleura on the opposite side, with the exception of not being quite so smooth: there was neither thickening nor the slightest increase of vascularity about this pleura. A large rent, from which the hæmorrhage had proceeded, was found in the substance of the lung.

Blood effused into joints presents at times appearances similar to those observed in the extravasations of blood into serous cavities, as described in the preceding cases.

In October 1844, in the knee-joints of a man who had been confined to his bed for a long time by paraplegia, I found flattened clots of blood lying in cupped surfaces on the cartilages of each patella; these clots were maintained in their situation by a thin transparent membrane perfectly smooth and polished, and at its margins bevelled off, gradually disappearing upon the polished surface of the cartilage, but the clots and their membranes were easily removed; the cartilage was blood-stained, and in various places presented a fibrous appearance, with deep clefts leading down to the bone. There was no increased vascularity about the joints, neither did they contain a larger quantity of synovia than usual.

Having thus proved that a membrane presenting the characters of a serous tissue, is formed from

blood extravasated, I shall now bring forward some cases of a newly-formed membrane being produced from the blood in the vessels themselves, and that too without any apparent signs of inflammation. The production of this membrane may take place either on the coats of the vessels or on the surfaces of clots contained in the vessels.

In June 1844, I examined an aneurismal pouch about the size of a large walnut, situated a little above the aortic valves. The arterial opening of this pouch, which was a free one, presented a thick, elevated ridge, deeply serrated and of a yellow colour, whilst its walls were remarkably thin and without a trace of yellow tissue; its internal surface was lined throughout by a fine transparent membrane, perfectly smooth and polished on its free surface, except at a few points where some small fibrinous coagula were adhering to it. This membrane and the lining membrane of the artery were, throughout the whole circumference of the aneurismal opening, perfectly continuous, and so beautifully adapted to each other, that, to the naked eye, they appeared to be identically of the same character.

In the preceding case it appears to me that the internal and middle coats of the artery had been destroyed, and that the pouch was formed by the cellular coat lined with a membrane which owed its origin to the fibrine of the blood constantly circulating through this abnormal cavity, and which had subsequently taken on the characters belonging to the serous coat of this vessel.

In June 1844, a man under the care of Mr. Hawkins was attacked with diffuse cellular inflammation of the inferior extremity, terminating in two days in extensive gangrene of the skin. In the superficial and common femoral veins of the affected extremity, I found extensive clots, not completely filling up the veins, but slightly adherent at different points, to their internal coats. These clots still retained in some places the colouring matter of the blood, whilst at others the colourless fibrine alone remained; in both the veins the clots were enveloped in a perfectly distinct, transparent, smooth, and polished membrane, presenting the appearance of a serous tissue. In the structure of these membranes were several distinct arborescent vessels, minutely injected; some of these vessels were of sufficient size to allow of the blood being made, by gentle pressure, to circulate through them: but no communication could be traced between these vessels and the coats of the veins. The membranes were easily peeled off from the surfaces of the clots with which they were in contact. The internal coats of the veins presented their natural colour, and polished surfaces, except at the points where the slight adhesions above-mentioned existed. The internal saphenic contained dark-coloured coagula, presenting the appearances of the blood commonly observed in veins.

In 1841 and 1844, I examined two large aneurisms of the aorta, which had been completely blocked up by fibrinous coagula, of long standing, and about two inches in depth. In each case, the

coagula presented, towards the cavity of the artery, a slightly-cupped surface, which was completely covered over by a fine, transparent, polished membrane, continuous with, and apparently exactly of the same nature as, the lining membrane of the artery. So perfect was the adaptation of these two membranes, that it was impossible, at first sight, to define the exact limits of each of them. After the removal of the coagula, the walls of the pouches were found to be remarkably thin, whilst the margins, at the arterial opening, of the size of a crown piece in one case, were elevated, remarkably thick, deeply serrated, and of a yellow colour.

With regard to these membranes, Mr. Bowman, some short time back, informed me of a curious fact. This gentleman, who has had an opportunity of examining, with the microscope, newly-formed membranes, similar to those described in the preceding cases of aneurism, told me that he has never yet found any epithelial cells on the smooth, polished surfaces of these membranes, although they, to the naked eye, appear to be exactly of the same nature as the membrane which lines the arteries.

The clots in the heart and large vessels of persons who have died a lingering death, sometimes present perfect types of the manner in which the extravasations of blood into serous cavities become encysted.

The upper surface of the clots, in the cavities of the heart, as this organ lies in its natural position, is not unfrequently covered over by a delicate, trans-

parent membrane, of a whitish colour, and perfectly smooth, and polished on its free surface, which may be easily peeled off from the blood, with which it is in contact. I have often observed this coating, even when all the other parts of the coagulum have been of a dark colour, and have traced it, wherever the blood has not been entangled, in the meshes of the fleshy fibres of the heart. In the large vessels, the internal surface of which is throughout perfectly smooth, the clots are oftentimes *completely* enveloped in a thickish membrane, of a character precisely similar to that described above, the other parts of the blood being sometimes of a dark colour, and firmly coagulated, except towards the centre of the clot, where the blood is more fluid. I have often peeled off this membrane, which is formed of coagulated fibrine, and which presents, after having been in water for a short time, exactly the appearance of the membrane, which is found enveloping some of the extravasations of blood in serous cavities.

Even blood extravasated into the tissues, becomes enveloped by a cyst, the formation of which may be, in some cases, traced to the fibrine of the extravasated blood.

In the beginning of 1839, a man was admitted into St. George's Hospital, with a severe injury of the thigh, and extensive extravasation of blood, in the subcutaneous cellular tissue, on the anterior surface of the tibia. After a few days, a great part of the blood over the tibia became collected into a

pouch, the margin of which was hard and circumscribed, the centre being soft and fluctuating. In the course of time, the pouch appeared gradually to contract, and its margins became less and less hard, until at length all traces of the extravasation of blood disappeared, and the parts resumed their natural appearance. All this took place without a single symptom of inflammation about the part; and yet the usual explanation given in such cases, is, that this pouch depends upon an exudation of lymph, poured out from the irritated and inflamed vessels in the neighbourhood. Is it not much more rational to suppose (when there are no symptoms of irritation, much less of inflammation present) that this circumscribing of the blood depends solely upon the fibrine of the blood already extravasated? Extravasations similar to the preceding one are often met with about the scalp, after an injury. In some of these cases, in which the blood has appeared to remain for a long time after the accident, a perfect cyst has been found, containing coagula and blood-tinged serum.

In the ovaries, I have had repeated opportunities of examining extravasations of blood, which are surrounded by a fine, delicate membrane, formed from the fibrine of the effused blood, the tissues in the neighbourhood being neither injected nor discoloured.

The well-known apoplectic cyst owes, I think, its origin, in many cases, to the fibrine of the blood already extravasated into the substance of the brain,

and not, as is commonly supposed, to an exudation of lymph, consequent upon irritation and inflammation of the neighbouring parts. A careful consideration of the various appearances observed in these cerebral apoplectic cysts will at once point out how strongly they resemble, in every point, the cysts which have been described as completely enclosing the extravasations of blood in the cavity of the arachnoid. This resemblance did not fail to attract the notice of Dr. Abercrombie,* who, in speaking of a large collapsed cyst found on the surface of the brain, expressly states that "it exactly resembled the substance which is found lining the apoplectic cysts in the structure of the brain."

Extravasations of blood into the cavity of the arachnoid have been found perfectly circumscribed by a false membrane, presenting all the characters of a serous tissue, six days after the first appearance of the symptoms. I have seen an extravasation of blood into the cavity of the pleura thus circumscribed five days after the accident. If the membrane is, as I suppose it to be in the majority of cases, formed by coagulated fibrine, there is no reason why these extravasations should not be found circumscribed, even at an earlier period; for I have already stated that I have repeatedly found the coagula of the large vessels, twenty-four hours after death, completely encysted in a membranous layer of coagulated fibrine.

* Loc. cit., p. 277.

By whatever process formed, these new membranes, in the cavities of serous tissues or in vessels, are so beautifully adapted to the original serous tissue, that it becomes a matter of the greatest nicety to trace the respective limits of each.

In the cavity of the arachnoid I have generally observed that the false membrane extends far beyond the limits of the pouch containing the blood: sometimes this membrane occupies the whole length of the parietal arachnoid, corresponding to the upper surface of one of the hemispheres, the pouch itself only measuring about the middle third of this space; in these cases the walls of the pouch are often one or two lines in thickness, the other parts of the membrane being pellucid, and gradually lost upon the arachnoid; these appearances are easily explained by the gradual addition of fibrine to the walls of the pouch, from the blood contained in its cavity.

Source of the hæmorrhage.—In speaking of exhalations of blood into serous cavities, Dr. Hodgkin states, "I know of no instance of the arachnoid containing blood, except from apoplexy, or injuries of the head;" but, at the present time, there are already on record many cases in which the hæmorrhage was not attributable to either of these causes, and recent investigations have, moreover, led many anatomists to the conclusion that exhalations of blood are found much more frequently in the arachnoid than in any other serous membrane. Mr. Ernest R[illegible]* states that, at the Salpétrière, six

* Loc. cit., p. 22.

of these cases were met with in seven months, and Mr. N. G. de Mussy met with eight of them in one year at the same Hospital; all these cases occurred in elderly persons. Several cases occurring in children have also been placed upon record by Messrs. Rilliet et Barthez.*

I have made use of the term "exhalation," as it is commonly used by the profession, both in England and on the Continent, to explain those cases in which the source of the hæmorrhage has remained undiscovered, however minute may have been the examination; but I agree with those anatomists who think that, in these so-called exhalations, the red blood disks always proceed from ruptured vessels, for the most part so minute as not to be discovered by our usual means of investigation. The extravasation, in these cases, generally takes place very slowly, and the blood is poured out in small quantities; sometimes the extravasation is intermittent, so that layers of coagulated blood, of various hues, have been found in contact with each other in the cavity of the arachnoid. The quantities of blood found in the cavity of this serous membrane, under these circumstances, have been very large; in Rostan's 91st case, already quoted, the cavity of the arachnoid contained more than two tumblers full of blood, with large dark clots.

These extravasations in the arachnoid are generally met with in cases where there has been a decided determination of blood to the head, by what-

* Traité des Maladies des Enfans, vol. ii. 1843.

soever cause produced. I have met with them in cases where there has been great anxiety of the mind, in poisoning by opium, in drunkards, in delirium accompanying phthisis, in maniacal patients, and in aged people, in whom I think that these effusions depend oftentimes upon the atheromatous deposit in the arteries of the brain and its membranes, which so frequently gives rise to extravasations of blood into the substance of that organ.

I shall avoid troubling the Society with a minute detail of the many symptoms which have been found to accompany these extravasations of blood into the cavity of the arachnoid; symptoms, which it is obvious must vary according to circumstances; such as, the greater or lesser quantity of blood extravasated; the rapidity with which the fluid is poured out, its situation, and its being spread out upon the surface of the brain, or accumulated in a cyst, &c., a due consideration of which circumstances will point out how hopeless must be, in most cases, any attempt at a correct diagnosis; and thus it is, that we find it stated by different authors that the cases of this nature which they have had an opportunity of watching, have borne the closest resemblance to diseases of the brain of totally opposite characters.

Connected with these extravasations there is however one remarkable circumstance, which having been already observed in several cases, ought not I think to pass unnoticed. I allude to an intermission in the symptoms either of coma, or even of paralysis. This intermission occurs under different circum-

stances: it may be dependent upon an interruption in the pouring out of the blood, during which time the brain gets accustomed to the pressure, and recovers its functions, until a further extravasation takes place: if carefully examined, the extravasated blood will in these cases be found of different hues, some of it being of a rusty colour, and other parts of the colour of recently-effused blood. This intermission in the symptoms also occurs in those cases where the extravasated blood has been completely surrounded by a perfectly organized cyst, such as in Case 5, in which the symptoms both of coma and of paralysis were observed to vary considerably: this is easily explained by the organization of the cyst, which, by pouring out a fresh quantity of blood or serum into its cavity, may produce symptoms of compression, which will vary according to the more or less rapid absorption of the fluid.

ON THE

COLOSTRUM OF THE COW.

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The Colostrum,—the new milk of the cow after calving,—differs, as it is well known, from ordinary cow's milk, in being of a richer yellow colour, less liquid, of greater specific gravity, and in possessing the property of coagulating when heated.

Resembling in the last-mentioned property the serum of the blood, or the substance of the egg, it has been supposed by some physiologists to be more animalized than common milk, and to contain even serum, and that its being coagulable by heat, is owing to the presence of serum.

This inference not having appeared to me to be proved in a satisfactory manner, and, *à priori*, not very probable, I have been induced to subject it to some trials, the results of which I shall briefly describe, tending to show, as I believe they do, that serum does not enter into the composition of the colostrum, and that its coagulability by heat de-

pends on a peculiar modification of its caseous portion.

The milk, the subject of the experiments I am about to describe,—selecting, as is desirable, a particular instance,—was from a healthy cow that calved for the first time on the 19th of July, and was drawn about an hour and a half after, the udder having begun to be distended about three weeks previously.

Its appearance under the microscope differed principally from that of common milk, in presenting larger oil globules,—a very few irregular flakes, probably epithelium scales,—a little granular matter, like curd, and a small number of granular corpuscles,—the granular bodies of the colostrum first described by Mandl, the largest of which were about $\frac{8}{4000}$ of an inch in diameter.*

It reddened, slightly, litmus paper. Its specific gravity, carefully ascertained before any separation of cream had taken place, was found to be 1075, and as high as 1080 after a portion of its cream had risen, and had been removed.

* The nature of these bodies seems to be somewhat doubtful,—whether they are aggregation globules, formed chiefly of albuminous matter,—or large oil globules, with particles of curd, or even of oil attached to their surface: from their apparent specific gravity, and some other circumstances, I am disposed to adopt the first opinion; when colostrum mixed with water is put by in a cool place, after a few days, when the greater part of the cream has collected at the surface, a sediment is found at the bottom, consisting chiefly of these globules.

To determine the degree of temperature at which it coagulated when heated, a portion of it, contained in a glass tube, was immersed in water, the temperature of which was gradually raised. At 160 degrees, Fahrenheit, it was unaffected; at 163 deg. it coagulated: the coagulum was rather soft,—but admitted of being inverted without flowing; it yielded readily to gentle pressure. It did not become hard when boiled, nor was its consistence materially increased by boiling. It may be deserving of mention, that the neutralization of the little free acid it contained by the addition of sesqui-carbonate of ammonia, had no marked effect on its coagulation by heat.

It was coagulated by rennet, and more readily than common milk. In a comparative experiment, whilst the latter required a temperature of 110 deg., the former underwent the change at the temperature of the atmosphere,—then about 65 deg. The coagulum of the colostrum was much softer than that of common milk, and the proportion of whey which separated from it was very much less.

Mixed with ordinary milk and heated, the colostrum acted like rennet; twelve measures of the colostrum mixed with forty-six of milk, formed a soft coagulum when the temperature of the mixture was raised nearly to the boiling point.

The colostrum was coagulated by all the acids that I tried on it, and this at ordinary temperatures, as the acetic, muriatic, nitric, and sulphuric; and also by the citric, tartaric, and oxalic, when mixed

with it in the state of powder. The coagulum it formed with the acetic and the mineral acids was soft, and of a grumous consistence; that which it formed with the solid vegetable acids was firm, like strong jelly, and diaphanous. When the mineral acids were added in large excess, the coagulum was dissolved, leaving the butteraceous part, which rose to the surface: ammonia, in the form of aqua ammoniæ, added largely in excess, formed a gelatinous mass of pretty firm consistence, which did not dissolve on immersion in water.

The colostrum left at rest underwent change slowly. After three days it still retained throughout its yellow hue: a part only of its cream had risen to the surface. After seven days, the lower part had become of the colour nearly of ordinary milk, retaining its liquidity, and having acquired increased facility of coagulating when heated,* whilst its upper part was of a richer hue, and less fluid, being covered with a thick pellicle of the consistence of cream-cheese. This had the peculiar smell of cream-

* In this respect resembling common milk, which after keeping a time, varying with the temperature of the air,—coagulates even below 160 deg.;—as to coagulability indeed presenting a complete gradation; when quite fresh, resisting it, as is well known, at the boiling point; and after keeping some time, coagulating at a low temperature, not exceeding that of the warm hand; in this state offering a subject for a pleasing experiment: it may be poured perfectly liquid into a thin glass tube, and merely by grasping the tube with the hand the effect is produced,—rapidly, almost in an instant, the liquid is converted into an apparent solid, and that without the disengagement of heat.

cheese, and was spotted with mildew, consisting of that kind of byssus which forms on cheese. After thirteen days, the inferior white portion had coagulated, having acquired the consistence with the properties of soft curd—the part above it having undergone little apparent change, that immediately over the curd retaining its yellowness or semifluidity, and the butteraceous cheese-like crust preserving the appearance before noticed, with increased firmness and increase of mildew.

Mixed with water (about two parts of water to one of colostrum), and agitated, and then allowed to remain at rest, cream rose to the surface, and curd formed and subsided, leaving, after about a week, a transparent or nearly transparent fluid between the supernatant cream and the sediment of curd. This fluid was acid, and held some curd in solution. It had a cheesy smell, was not rendered turbid by boiling, nor by acetic or the citric acid, but yielded a precipitate with the three mineral acids; and when kept, a white crust formed on its surface. The formation of this crust was accompanied by a disagreeable smell, similar to that of decaying cheese, but rather more offensive, partaking of the putrid odour, owing probably to the presence of some granular corpuscles. When the crust was separated by filtration, a fresh one formed on the fluid in a few days, and this successively for many days. The crust, or pellicle, consisted chiefly of little cylindrical masses, rounded at their extremities, about $\frac{1}{1000}$ of an inch in length, by about $\frac{1}{2000}$ in width. After

the fluid had ceased to yield this pellicle, it was rendered turbid by nitric acid, as is also the whey of ordinary milk, whether coagulated by rennet, or spontaneously on its becoming sour by the absorption of oxygen.

Other specimens of colostrum which I have examined have afforded similar results. I may notice two in particular, the specific gravities of which were ascertained with care, and the degree of temperature at which they coagulated. The one was from a cow that calved in September, and was drawn almost immediately after: its specific gravity was found to be 1070. Whilst still warm, it reddened litmus. It was pretty firmly coagulated at 160 deg.; at 150 deg. it formed a soft coagulum; and at 140 deg. it became thicker. At the lower temperatures, a much longer time was required for the effect to be produced than at the higher. The other specimen was from a cow that calved in December, and was drawn about a quarter of an hour after. It was less thick than the colostrum usually is, was of specific gravity 1057, and coagulated at about 162 deg.

What are the inferences to be drawn from the preceding experiments? Do they not show that the peculiarities of the colostrum are not dependent on the presence of serum, and, in brief, that the colostrum is destitute of serum?—The effect of rennet on it, which I find does not coagulate serum,* the

* See Researches Physiol. and Anat, ii. p. 97; the experiments related there have been repeated with the same negative result.

effect of the vegetable acids on it, and the changes which it undergoes on keeping, when partially exposed to the action of the atmosphere, are the facts most to be insisted on. And, in corroboration, I may mention the results of trials on the heating of mixtures of common milk and serum in different proportions. Serum, in certain proportions, like the white of egg, I find does occasion the coagulation of milk,—giving rise to custard ; but it requires a pretty large proportion : five parts of serum with fifty-five of milk, even when boiled, had no effect ; and even when the former was increased to ten, and the latter reduced to fifty, the mixture did not coagulate till its temperature was raised to about 195.

Whilst all these results are opposed to the idea that serum forms a part of new milk, they favour the conclusion, that the caseous portion of the colostrum is in a state somewhat different from its ordinary condition in the milk of the cow, modified in some manner, either in consequence, it may be, of a slight difference in composition, or owing to the influence of the other ingredients of the milk with which it is mixed, especially the granular bodies, if admitted as aggregation corpuscles, which may perform the part of rennet, in promoting its coagulation, and the butteraceous ingredient, more abundant in it than in common milk, separating more slowly, and which must protect it more powerfully from the action of the atmospheric air, and from the changes consequent on the absorption of oxygen. And, that the caseous portion of milk

does admit of modification, and that not inconsiderable, is shown in a remarkable manner, on comparing it, as existing in the human milk and in cow's milk; in the latter, even in its ordinary condition so readily coagulable, whilst in the former it resists this change, whether acted on by acids or rennet, and yet is easily obtained by means of evaporation, when freely exposed to the air, and without evaporation from the slow and long-continued action of atmospheric air.*

Physiologically considered, the most marked circumstances belonging to the colostrum, are the concentration of nutritive matter in it; the greater facility of its coagulation by rennet, compared with older milk, and its greater power of resisting change when exposed to the action of atmospheric air. These are qualities which may be eminently serviceable, viewing it as the first food of the young animal. Its easy coagulation may suit it to the stomach, in which probably the gastric juice at first, is in small quantity and feeble. Its power of resist-

* A portion of milk from a healthy young woman, five weeks after delivery, of sp. gr. 1033, kept in a stoppered vial, to which air had access, a thin slip of paper having been placed between the stopper and its neck, and kept in a dark place, of about 55 deg. temperature, from November till May, had lost its milk-white hue, and deposited curd as well as thrown up cream. Strained, the whey was found of sp. gr. 1020 : 8·1 grains of dried curd were obtained from 312 grains of milk. The whey reddened litmus paper, and was not coagulated either by boiling, or by nitric acid. The curd was very like that which constitutes the chief portion of the alvine evacuation of infants at the breast.

ing change, and remaining semi-fluid, may adapt a part of it to the intestines, to promote the removal of the meconium. Whilst its concentration as nutritive matter may fit it to perform for the calf the same part that the substance of the egg serves which enters the intestine during the latter stage of fœtal development in the instance of birds, reptiles and fishes.

The change which takes place in the cow's milk, watching it after calving, is not unfavourable to this view of the uses of the colostrum. Recurring to the first example, the milk of the cow which calved in July, that drawn after 24 hours, was of sp. gr. 1039; was less thick and less coloured, and coagulated when heated to about 180 deg. The milk of the following morning, of sp. gr. 1038, did not coagulate even when boiled, subjecting it to heat five hours after it was drawn; but after having been kept thirty hours, then it coagulated like the preceding, at about 180 deg. Milk of the following morning, drawn on the 21st July, about 60 hours after calving, was of sp. gr. 1033, differed hardly perceptibly from common milk in appearance, and did not coagulate on boiling, even after having been kept about fifty-four hours, at a temperature little below 70 deg., and by day amounting to that.

If the special use of the colostrum of the cow is such as I have inferred, it may be expected that the first milk of other animals will be found to be similar in its properties,—at least, of all those the young of which, like the calf, are born fully formed and

vigorous, with good use of their limbs almost immediately after birth. And so far as I have been able to learn, this is so. The new milk of the ewe, of the mare, and of the sow, I am informed by intelligent farmers, is as rich and thick, and, in the instance of the two first, coagulates when heated. But whether the milk of the sow has the same property, I have not been able to ascertain.* I may mention another instance—it is the milk of the giraffe. My friend Mr. Gulliver informs me that a small quantity "drawn within an hour after the young one was born, (May 20th, 1841,) was excessively rich in fat-like cream, not rendered clearer by any quantity of ether, and became thicker or clotted in a watch glass over a candle."

Whether the first milk of those animals, the young of which are born helpless and feeble, as of the carnivora, is also like the preceding, cannot, that I am aware of, be at present determined, for want of facts. I am disposed, however, to conjecture that it is similar. This conclusion is founded on analogy, having found that the first milk of the bitch is so,—coagulating when heated, and yielding a large proportion of animal matter (35·2 per cent.)

* Since the above was written, I have examined the first secreted milk of the ewe and sow, and have found them very like that of the cow. The ewe's was of sp. gr. 1060, reddened, slightly, litmus, coagulated at about 165 deg., and yielded, when evaporated to dryness, 35·7 per cent. solid matter. The sow's was of sp. gr. 1057, and coagulated at 160. Milk from another sow, the first drawn also, was found of sp. gr. 1062.

when evaporated to dryness. I could not procure sufficient to determine its specific gravity. In the instance of the carnivora, it may be requisite for the first milk to be rich, from the manner in which these animals feed, having to leave their young to procure food, and to be absent, it may be, an uncertain time. And, in accordance with this, is the fact, that the human milk, the first drawn, is not unusually rich, and does not coagulate when heated; at least these are the results of the few trials of it that I have made. The milk of a healthy young woman, who had been confined five weeks, nursing her first child, I found of the sp. gr. 1033; it yielded on evaporation 10·22 per cent. of solid matter. The first milk of a healthy woman, ætat. 40, after giving birth to her sixteenth child, was of sp. gr. 1033, and yielded on evaporation 12 per cent. solid matter. The first milk of another woman, of whose age or condition I omitted to make a note, was of sp. gr. 1022. Examined four days after, this woman's milk was found to be of sp. gr. 1035, and to yield on evaporation 11·6 per cent. solid matter. This peculiarly dilute state of the human colostrum, if proved to be general, seems equally suitable to the condition of the offspring and mother, the one helpless and feeble, not requiring concentrated nourishment, the other commonly, from a certain degree of exhaustion during labour, ill fitted to yield such a supply;—and, moreover, being designed for domestic life and support, and not under the necessity of separating herself from her offspring to

go in quest of precarious food; offering in this point of view, another instance to the vast number of examples of harmonious adaptation; and it may be also one circumstance more, by which man as an animal is distinguished from every other.

CASE

OF

OBSTRUCTION OF THE LARGE INTESTINE,

IN WHICH

THE ASCENDING COLON WAS OPENED WITH SUCCESS:

THE PATIENT DYING THREE MONTHS AFTERWARDS OF ANOTHER DISEASE.

By SAMUEL EVANS, Esq.,

OF DERBY.

Communicated by W. BOWMAN, F.R.S.,

ASSISTANT-SURGEON TO KING'S COLLEGE HOSPITAL.

Received January 4th—Read April 12th, 1845.

Lewis Street, ætat. 23 years, a farmer of robust frame, has for several years been liable to occasional attacks of diarrhœa, and when he was seven years old, was much troubled with tape-worms, which distressed him for many months.

In September 1843, he ate a large quantity of sloes, and was shortly afterwards seized with violent pain in the bowels, resembling colic, which lasted for thirteen hours; his medical attendant bled him, and administered active aperients. After several copious evacuations he became easier, and in a few days he resumed his usual employment, which he followed without interruption till the middle of January.

About the third week in January the attacks of

colic recurred, and were succeeded by dark-coloured and fœtid watery stools, mixed with scybala; the attacks became more severe on the 5th of February, and I saw him for the first time on the 7th. He was suffering from severe pains recurring at intervals of five or ten minutes; the abdomen was greatly distended, but free from tenderness on pressure. The right iliac region appeared to stand out in relief from the distended abdomen; the pains were principally felt in this situation, but extended to the right loin, and to all parts of the abdomen: he had not had any evacuation from the bowels since the 5th, notwithstanding the administration of the most active aperients prior to my visit—pulse 76, soft—tongue moist and clean, skin cool and perspiring.

℞. Liq. opii sedat. ♏ xl.
Aq. menthæ ʒiſs ft. Haustus statim sumendus.

℞. Extract colocynth. c. gr. v.
Ol. Tiglii ♏ i. ft. Pilula. 3^{tiis} horis sumenda.
Adhib^{r} Enema colocynth. 4ta quaque hora.

February 8th.—The opiate afforded much relief, but after six hours the pains increased, and they have since recurred with great severity at intervals, followed by distressing sickness and vomiting of bile and mucus. The croton oil appearing to increase the pain and sickness, has been discontinued.

℞. Opii puri gr. ii.
Hydrarg. chloridi gr. viii. ft. bolus statim sumendus.
Sumat quaque 4ta hora, vel pro re nata Pil. sapon. comp. gr. v.

A soap-and-water injection to be given every three hours.

9th.—He has not vomited since the first dose of the opium, but has had no relief from the bowels: pulse 76, soft and compressible.

An elastic tube was passed to the extent of eighteen inches, and about three pints of soap-and-water were injected, which returned in a few minutes with very small portions of fæcal matter.

From the 9th to the 12th there was but little variation in the symptoms; the treatment consisted in the administration of opiates and castor oil, with enemata of castor oil and turpentine, but no fæcal evacuations were obtained.

12th.—I was requested to see him in the night. I found him vomiting large quantities of olive-coloured stercoraceous matter, and suffering from most agonizing pains in the abdomen; he attributed this attack of pain and vomiting to a dose of castor oil, which had been taken a short time previously. One hundred minims of liq. opii sed. were given, which relieved him in half an hour; and when he was sufficiently composed, the rectum tube was again passed, but without affording any relief.

13th.—Has slept comfortably for several hours, and has not suffered from any severe paroxysms of pain since he took the opiate. Several small portions of fæcal matter, and a large volume of flatus, were brought away by a soap-and-water injection administered this morning, after which the distention of the abdomen was diminished.

From this time to the beginning of April, the size of the belly gradually increased: he also daily suffered many paroxysms of pain; and although at intervals of one, two, or three days, large quantities of flatus, and a few very small portions of clay-coloured fæces escaped, it was quite obvious that there still existed a partial obstruction in the bowels, since the distention of the abdomen, and the *swelling in the right iliac region*, had considerably increased.

During this protracted illness he was seen by Dr. Bent of Derby, and several of my medical friends, by whom various remedies were proposed, but he obtained relief *only*, from the administration of narcotics. The patient had become much emaciated, was extremely weak, and his general health greatly impaired; the vomiting recurred almost daily, and the stomach could retain only the smallest quantities of food; the lower extremities were much swollen, the tongue and skin dry, the pulse frequent and small.

On the 25th March I represented to the sufferer, that relief might probably be afforded by the formation of an artificial anus in the loin by the method of Callisen, as modified by Amussat. He was desirous that the operation should be performed immediately, but he yielded to the wishes of his friends to postpone it for a time.

April 8th.—He has become very much emaciated; the abdomen is distended to the greatest possible degree; the convolutions of the small intestines, which appear to be greatly enlarged, can be clearly

traced through the attenuated coverings, and their vermicular motions distinctly observed. The swelling in the right iliac region has so much increased as to present the appearance of great deformity. The integuments in this region are extremely thin, and of a livid colour: there is considerable œdema of both legs; tongue dry and fissured; the mucous membrane of the tongue and of the mouth is covered with an aphthous eruption; he has not retained the smallest quantity of food, and has frequently, since the 4th inst., vomited dark, watery, bilious matter; there has not been the slightest appearance of fæcal evacuation for four days: pulse feeble and fluttering, varying from 112 to 124: respiration 16: urine scanty and turbid, of a dark olive colour, and fœtid.

The inefficacy of all other measures having become apparent to the patient's friends, they were now most anxious that the operation, already proposed, should be performed, being convinced that unless relief could be speedily obtained, death must in a few days terminate his sufferings. It was therefore arranged for the next day, when, in the presence of my friends, Mr. Nicholson, Mr. Walker, and my brother, Mr. D. Evans, I proceeded as follows:—

The patient having been laid on the bed with his face downwards, two pillows were placed under the belly for the purpose of rendering the right loin more prominent. I made an incision of four inches across the loin, commencing it at the outer margin

of the sacro-lumbalis muscle, about an inch above the crest of the ilium, and extending it forwards in a direction parallel to the crest. Having divided the latissimus dorsi, and the outer margin of the quadratus lumborum muscles, I proceeded to expose the bowel by opening the fascia; but in doing so, I also opened the intestine, which was intimately united with the fascia, by dense cellular tissue, without any intervening fat: indeed, no fat whatever was brought into view, as I had been led to expect there would be—a fact that may be accounted for by supposing the extreme distention of the head and ascending portion of the colon, to have pressed upwards the kidney with its circumjacent fat, above the line of incision. Possibly, however, this fat may have been absorbed, as the man was extremely emaciated. The instant the bowel was punctured, a profuse quantity of semi-fluid, clay-coloured fæces was projected with great force through the opening, and continued to escape for a considerable time, so as to amount altogether to more than two gallons. I then enlarged the orifice in the intestine to the extent of one inch in a transverse direction, or parallel to the external incision. The last steps of the operation consisted in connecting the edges of the incision in the intestines, with those of the outer or anterior half of the external wound, by means of five sutures, the inner third of the external wound being closed by a pin, and twisted suture, and covered by a few strips of adhesive plaster. A small piece of oiled lint was then placed between the

edges of the wound in the intestine, and cloths wetted with warm water were laid over the wound. The abdomen, now reduced to its natural size, was supported by a many-tailed bandage, and the patient was placed on his right side in bed, so as to allow of a free escape of the contents of the bowel through the wound.

9 P.M.—He has had no return of vomiting, the sense of distention had ceased, and he feels in every respect greatly relieved: he complains of a smarting pain in the wound, which extends to the right iliac region; pulse soft, regular, 110. Ordered 40 drops of liq. opii sedativus.

April 10th.—The pains in the wound and right iliac region were relieved by the opiate: he has had six hours of undisturbed sleep, and he feels much refreshed this morning; is quite free from pain, but complains of extreme weakness, great exhaustion, and craving for food; pulse 100, feeble; tongue moist, covered with aphthæ. The bandage having been much soiled by the discharge from the wound, was removed and replaced by a napkin; the lips of the wound were tender, but not swollen; a poultice of bread-and-water was applied.

Gruel with small quantities of brandy to be taken frequently.

11th.—10 A.M.—He is much exhausted, complains of oppression, slight difficulty of breathing, and a sense of sinking at the epigastrium, has passed urine every three or four hours with great pain in the glans penis; there has been a continual discharge

of liquid fæcal matter from the wound, which he compares to the working of yeast from a beer barrel.

℞. Liq. opii sedativ. ♏ xl.
Aquæ menthæ ʒi. ft. Haustus statim sumendus.

℞. Tinct. opii ʒi.
Tinct. catechu. ʒfs
Confect. aromat. ʒi.
Decocti cinchonæ ʒviifs
M. ft. Mistura sumat ʒi. quaque 4ta hora.
Beef tea, milk and eggs.—Small quantities of wine occasionally.

April 12th.—12½ A.M.—Has passed a restless night, the difficulty of breathing has increased; he has been very much troubled with an accumulation of frothy mucus in the trachea; respiration short and quick, 48 in the minute, and on the slightest exertion it is raised to 55; he frequently makes an ineffectual effort to cough; pulse 112, very weak; the bowels have been freely relieved through the wound since yesterday; urine is passed more freely, but still with great pain.

He complains more than ever of the sense of sinking and emptiness at the epigastrium, and says he cannot exist without some solid food: he was accordingly ordered to take two mutton chops daily, and to continue the wine and milk as before.

Rep. Mist. cinchonæ.

Rep. Haust. opii.

14th.—Has continued to improve since the 12th. Has had light yellow evacuations through the wound daily; urine healthy, and voided without pain; pulse

80; respiration 36. His emaciation is extreme, and has much increased since the operation; the aphthous state of the tongue and œdema of the legs have quite disappeared; he breathes more easily, and the mucous rattle is considerably diminished: he has almost lost the feeling of emptiness and sinking at the epigastrium; tongue clean; passes urine more easily, but still with slight pain.

18th.—His improvement has been progressive; his general health is much improved; wound healthy and granulating; bowels relieved daily through the artificial anus; the evacuations are of a light yellow colour; they are liquid, and contain small particles of imperfectly-digested food.

A long tube was passed up the rectum, and three pints of water injected, which returned in a few minutes, containing numerous small particles of fæcal matter.

Rep. Mistura et Haust. opii.

22nd.—Continues to improve in every respect.

26th.—Since the 24th there has been a continual oozing of a thin serous discharge of a pale colour from the wound, so abundant as to saturate many folds of linen in the course of the day, and for the same period he has complained of sharp pricking pains in the right hypochondrium, extending towards the cæcum, and occurring in paroxysms three or four times a-day.

There have been free evacuations daily from the artificial anus since the 22nd; the consistence of these is thin, they are of a yellow colour, and of a

fæcal odour; he feels extremely weak; his pulse is 100, rather small and compressible.

The posterior part of the wound which was brought together with the pin and twisted suture has healed; the wound in the intestine is granulating and in a healthy state.

℞. Misturæ cretæ comp. ℥vii.
Tinct. catechu. ℥ſs.
Tinct. opii ℥ſs.
M. ft. Mistura sumat ℥i. ter die.

℞. Opii purificati gr. i.
Pulv. Kino gr. v.
Ft. Bolus omni nocte sumendus.

29th.—The yellow serous discharge mentioned in the last report, continued to escape without any abatement, until yesterday: he has had copious fæcal evacuations through the opening, daily; complains of increasing weakness, has less appetite, slight nausea and frequent hiccough; pulse 110.

Rep. Pil. opii c̄ Kino mane nocteque, et Mistura cretæ.

The wound was plugged with lint dipped in strong solution of alum.

May 6th.—The sharp pricking pains have ceased, the serous discharge has also diminished daily, and is now quite gone. He is gaining flesh, his appetite is good, pulse 94; the wound in the intestine is now perfectly united to the skin, but the orifice is so reduced in size as barely to admit a rectum tube of common dimensions. When this was passed about five inches up the ascending colon, it seemed

to meet with an obstacle, and as it caused pain it was immediately withdrawn. The evacuations escape entirely by the artificial anus, to which a plug is adapted.

As he feels no natural desire to defecate, he finds it necessary to remove the plug from the artificial anus four or five times a-day, whilst he is in the horizontal posture, and the fæces being always semi-fluid, easily escape into a vessel placed under the orifice. Injections of warm water have been administered by the rectum every two or three days, but they have invariably returned mixed with small portions of opaque mucus.

June 8th.—Absence from home has prevented my seeing the patient during the past month: his convalescence during that time has been uninterrupted; the rapidity with which he has gained flesh is remarkable; for the last fortnight he has taken daily exercise. He still complains of uneasiness in the right hypochondrium, aggravated by exercise, though he is able to walk several miles.

June 18th.—He has become quite fat, feels in good health and spirits, and there appears every prospect that he will soon regain his accustomed strength. The evacuations continue to take place entirely by the artificial channel.

June 25th.—I found him suffering from symptoms of indigestion attended with feverish excitement: has had copious evacuations from the orifice, during the last 24 hours, of an olive green colour, and offensive smell; tongue dry, pulse full, 110. He

ascribes this attack to a hearty dinner of ham and peas which he took yesterday.

Rep. Mistura cretæ ℥ Tinct. catechu. 6ta quaque hora.

℞. Hydrarg. ℥ Creta.

Pulv. Ipecacuan. c. ää gr. v. ft. Pulvis mane nocteque sumendus.

June 27th.—The diarrhœa has considerably abated; the motions have recovered their usual yellow colour, complains much of thirst, tongue dry and red, pulse 98, skin cooler, but not moist: states that for nearly a week he has voided a large quantity of urine, and of a paler colour than usual.

June 28th.—Seven quarts of pale straw-coloured urine, having all the sensible properties of saccharine urine, and of sp. gr. 1040, have been passed during the 24 hours: thirst abated, pulse 100. The patient declares that the thirst, diuresis, &c., had not existed more than a week, and this assertion is fully confirmed by his mother, whose anxious watchfulness rendered her quite alive to the slightest variation in the symptoms. According to her statement, the urine had seldom exceeded 3 or 4 pints daily, and was of the colour of pale amber.

℞. Acidi nitrici diluti ʒifs.

Tinct. opii ʒifs.

Decocti cinchonæ ℥viii. m ft. Mistura sumat ʒi. ter quotidie.

Rep. Pil. Opii et Kino mane nocteque.

Diet—bread, beef, and mutton; and to drink beef-tea and toast-water.

To avoid sweet, acid, and spirituous drinks; also vegetables and fruits.

July 1st.—Has passed four quarts of urine in 24 hours, has less thirst, and less craving for food; pulse 90.

July 4th.—At 2 P.M. I was hastily summoned to visit him, and was surprised to find him in a state of extreme prostration, suffering most agonizing pain in every part of the bowels, frequent sickness, and vomiting of dark grumous matter. The abdomen is slightly swollen, tympanitic, and extremely tender; he is very restless, and continually changing his posture; his pulse is feeble and fluttering, 130; extremities cold and livid; expression of countenance, and every symptom, indicate approaching dissolution.

Upon inquiry, I ascertained that on the 2nd inst., after riding a distance of six miles in an uneasy cart, he was extremely fatigued, and shortly afterwards inflammatory symptoms supervened; but hoping that after a little rest these symptoms would abate, he deferred sending for me.

5th.—Died this morning at 8 o'clock.

6th.—*Examination of the body* 23 *hours after death.*—The body presented an average development of muscular and adipose tissues.

The artificial anus was a small constricted circular aperture, about $\frac{5}{8}$ths of an inch in diameter; its margin was firm and unyielding; the integuments near the orifice were slightly drawn inwards, and

their connection with the coats of the intestine were so intimate, that the line of union of the tissues could not be defined.

The abdomen contained 6 oz. of serum, of a dirty yellow colour.

The stomach and small intestines were greatly distended with flatus; the peritoneal surface of the lower two thirds of the ilium was highly vascular, and of a florid red colour.

The mucous membrane of the stomach and small intestines appeared healthy; the latter contained a small quantity of pale yellow chymous matter.

The cæcum was enormously distended, and was nearly as large as a stomach of ordinary size; the ascending colon was also much enlarged.

About a quarter of an inch from the commencement of the transverse colon, that is, just beyond the angle formed by the junction of the ascending and transverse portions of the colon, the gut was contracted to about three-quarters of an inch in diameter, and about the same in length; the contracted portion presented a cartilaginous hardness.

The serous covering of the cæcum, ascending colon, and the first half of the transverse colon, was highly vascular; there were numerous small patches on its surface, of a deep red colour, which were partially covered with recently-effused lymph.

The anterior three-fifths of the circumference of the first half of the ascending colon were bounded by peritoneum, and the posterior two-fifths by the

fascia covering the lumbar muscles; and it was in the latter part of the bowel, about half an inch beyond the cæcum, that the artificial anus was formed.

About 2 oz. of dark brown fæcal matter were found in the cæcum and ascending colon; the mucous membrane was of a deep claret colour, and thickly covered with bloody mucus.

The contracted portion of the colon was almost as hard as cartilage; it formed a ring about three lines thick, and nine lines broad, and appeared to consist of compact white fibres, arranged irregularly around the intestine; between the mucous and muscular coats the stricture would just admit a crow-quill; its inner surface was in a state of ulceration, and presented an irregular granulated appearance without any traces of mucous membrane. The mucous membrane of the first third of the transverse colon was in a state of uniform injection; the whole extent of the transverse colon was lined with a muco-purulent secretion, of a greenish colour, which was most abundant near the stricture.

The descending and sigmoid portions of the colon were quite empty and contracted, and presented no morbid appearance.

Both kidneys were larger than natural, their structure was not altered, but the cortical substance was rather paler than natural.

Liver, spleen, and pancreas, healthy.

Thorax.—The pericardium was adherent to the heart in its whole extent.

The heart was considerably enlarged.

Both ventricles were dilated, and the walls of the left ventricle thicker than natural; the valves and lining membrane of the heart appeared healthy.

Right lung natural.

The inferior halves of the pulmonary and costal surfaces of the left pleura were connected by long cellular bands. Old cellular adhesions existed on the lower half of the left lung, which was condensed, and of a deep purple colour, apparently from infiltration of blood and serum.

Remarks.—This is the eleventh case on record, in which Callisen's operation (modified by Amussat) has been performed in the adult, in consequence of obstruction in the intestinal canal.

From the previous history of the case, it would appear that the disease had been of slow progress, and of considerable duration. The gradual distention of the abdomen, and the absence of any satisfactory evacuation from the bowels during a period of seventy days, served to give strength to the opinion, that there existed a progressively increasing obstruction in the bowels, which, from the small amount of fæcal matter evacuated during that period, approached very nearly to a case of complete obstruction. The operation was suggested to the patient, on the 25th of March, as the only measure calculated to afford relief; but in consequence of the interference of his friends, it was not performed until the 9th of April, when the extreme emaciation, the great prostration of strength, the aphthous state of the mouth and fauces, and the œdema of the legs,

indicated the alarming condition of the patient; and it is impossible to imagine a case more unfavourable for the performance of the operation.

The enormous discharge of two gallons of fæcal matter through the artificial anus, serves to prove the degree of distention of which the cæcum is capable.

The patient's improvement was very gradual; but he was so much recovered in health in two months after the operation, that he was enabled to walk several miles.

On the 18th of June he was reported to have become quite fat, and at that time there appeared to be every prospect of his restoration to health. But our hopes were disappointed by the imprudence of the patient in regard to diet and exercise.

On the 25th of June I found him suffering from diarrhœa, with symptoms of dyspepsia: on the 28th a diabetic state of the urine was observed, and on the 2nd of July symptoms of peritoneal inflammation supervened, which appear to have been the immediate cause of death. As far as the operation is concerned, the case was successful; and if the patient had possessed a sound constitution, there is reason to believe that he might have enjoyed a tolerable share of health, but subject to the inconvenience attending an artificial anus, as the obstruction of the bowels was so complete as to preclude all hope that the continuity of the canal might ultimately be re-established.

ON THE

MORTALITY IN PRISONS,

AND THE

DISEASES MOST FREQUENTLY FATAL TO PRISONERS.

By WILLIAM BALY, M.D.,

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Received November 9th, 1844—Read February 25th, 1845.

The amelioration of public health, which has been effected by the diffusion and practical application of scientific truths, is one of the most remarkable features in the history of society during the last sixty or seventy years. This happy result of the advance of civilization is manifested in the improved condition of all classes of the community, but is seen nowhere more conspicuously than in the gaols and houses of correction in this country.

In Howard's time, the prisons of England, like those of the rest of Europe, were so filthy, crowded, and ill ventilated, that the air which the unfortunate inmates were compelled to breathe had an insupportable odour, and was almost constantly laden with the infection of putrid fever. The most benevolent persons scarcely ever ventured within the

walls of these noisome hotbeds of vice and disease; and even Howard found it necessary to protect himself against infection by carrying about him vinegar, and other reputed antidotes. This state of things no longer exists. The prisons of England are now, for the most part, remarkably clean, and if not *perfectly* ventilated, are very rarely so crowded as to endanger life by the development and propagation of typhus or putrid fever. To casual observers, indeed, they may appear to have every requisite for the preservation of health, which is consistent with the state of imprisonment; and by all who witness their present condition, they must be admitted to offer a striking contrast to the gaols visited and described by the great English philanthropist.

But, notwithstanding their greatly improved condition, an examination of the statistics of many of these establishments shows that the rate of mortality is much higher amongst the prisoners confined in them than amongst persons of the same age in society at large. This fact, which has not hitherto occupied much of the attention of members of our profession in England, is, I think, well worthy of their consideration; for, setting aside the possibility of aiding in the further improvement of the health of prisons, some facts of direct professional interest, and a few perhaps capable of useful application in the field of practical medicine, may be gathered from an inquiry into the causes which have produced the unusual amount of disease, and

high rate of mortality amongst incarcerated criminals. There are several reasons why this inquiry has been so much neglected. The principal one, however, seems to be, that the disease which is now chiefly fatal to prisoners is chronic in its growth and progress, requiring many months for its complete development; while the opportunity of observing the effects of imprisonment during so long a period has been afforded to few medical men in this country. In the county gaols and houses of correction of England, the average time during which criminals are confined does not exceed a few weeks. In the hulks, the average time of imprisonment has been much longer; but these establishments have never attracted much of public attention, and the state of health in them has not been made the subject of inquiry. With respect to the only other penal establishment, the Millbank Penitentiary, where criminals were confined for periods of two, three, or four years, there has been no want of publicity or of investigation; but here another circumstance has prevented inquirers from examining the effects of imprisonment on the health. A severe epidemic having once prevailed in the Penitentiary, and having been ascribed by the medical men who witnessed it, to a local noxious influence, no other cause seemed requisite to explain any amount of disease which might subsequently prevail. Hence, of late years, the nature of the maladies most generally fatal to the prisoners in the Penitentiary has, I believe, been seldom inquired after, and has be-

come known to few persons besides the medical officers of the institution.

Personal observation of the health of the prisoners in the Penitentiary during the year 1840 and subsequent years, and an examination of the medical records of the establishment, convinced me that a great part of the most serious diseases there prevalent, and especially of such as proved fatal, arose, not from any noxious influence peculiar to the locality, but from causes which were likely to be common to most if not all prisons. The desire of arriving at a satisfactory conclusion as to the relative activity of different causes of disease in the Penitentiary, led me to institute a rather extensive inquiry respecting the rate of mortality, the nature of the more prevalent fatal diseases and their causes in other penal establishments.

The main results of this investigation were, first, that imprisonment, continued for periods of 2, 3 or 4 years, produced everywhere a high rate of mortality; and secondly, that, although in particular instances other causes might contribute to increase the number of deaths, yet, in all prisons, the Millbank Penitentiary included, the increased mortality was chiefly due to the prevalence of *one and the same disease*, namely, tubercular scrofula. The facts upon which these conclusions are based, will be detailed in the paper now before the Society.

The present seems to be a favourable period for reviewing the medical history of prisons, especially in this country. For within the last two or three

years great changes have been effected in the whole system of secondary punishments, and in the state of prisons. 1st. The prison at Millbank has, since June 1843, ceased to exist as a Penitentiary, having been converted into a great central depôt for convicts destined for transportation. The opportunity of observing the effects of long imprisonment in this establishment is therefore now at an end.

2nd. The diet of prisoners in all gaols throughout England has been regulated according to an uniform and improved scale promulgated by the Secretary of State.

3rd. New prisons have been erected in which advantage has been taken of the most recent improvements in practical science, in order to render the system of warming and ventilation as perfect as possible. In these prisons, and also to a less extent in many others, great care is now exercised to afford varied employment for the minds and bodies of the prisoners, and, in a word, every means is used to maintain as perfect a state of health as is consistent with the condition of restraint, and with the discipline which holds out the fairest promise of reforming the moral character.

These changes constitute a new epoch in the history of prison discipline in this country. Some years however must elapse before the effects produced on the health by confinement in the newly-erected prisons, and under the new arrangements, can be satisfactorily determined. The remarks in this paper refer only to the results of imprisonment during the

period of fifteen or twenty years just past, and under the conditions which during that period have been presented by the Millbank Penitentiary, and by most other great prisons in Europe and America.

The plan which I shall follow, is first to determine the rate of mortality in the Millbank Penitentiary and in other prisons, comparing the mortality in each prison with that of the free population in the country to which it belongs; secondly, to point out several causes which influence very much the number of deaths in prisons, and occasion the rates of mortality within them to vary quite independently of any difference in the system of discipline, diet, and other internal arrangements; and lastly, to show the relative prevalence of different classes of fatal diseases amongst prisoners, and to demonstrate the influence of the duration of imprisonment, diet, season of the year, age, sex, and other causes, on the production and frequency of those diseases to which the mortality of prisoners is chiefly due.

In these several divisions of the subject, I shall generally, first examine the data afforded by the Millbank Penitentiary, and afterwards compare these data with those derived from other prisons.

I.—Rate of Mortality in Prisons.

Rate of Mortality in the Millbank Penitentiary.—When we endeavour to determine what has been the rate of mortality in the Millbank Penitentiary, we meet with a serious difficulty, owing to the

humane practice pursued by the superintending committee, of recommending to the mercy of the Crown all such prisoners as appeared to the medical officers to be sinking under the effects of imprisonment, or at least to be already in a much impaired state of health.*

The total number of deaths during the eighteen years from the commencement of 1825 to the end of 1842† was 205; the average number of prisoners during the same period was 532 (see Table I. in the Appendix). The average annual mortality, therefore, was only a little more than twenty per thousand (21·380), or two per cent. But besides the prisoners who died in the Penitentiary, there were 355 invalided, or pardoned on medical grounds,

* The rule which guided the medical officers in this matter was as follows:—"If any prisoner shall be afflicted with any disorder in which change of air shall appear to be necessary to preserve his or her life, or from which the patient is not likely to be sufficiently recovered to quit the infirmary during the term for which he or she shall have been sentenced to confinement in the Penitentiary, the medical superintendent shall state the circumstances of the case to the committee, in order that they may consider of the propriety of bringing such prisoner under the notice of the Secretary of State, as a fit object of the royal mercy."

† In consequence of the great prevalence of disease in the Penitentiary during 1823, the institution was in that year closed for several months. It was re-opened in October 1824, and was finally converted into a depôt for convicts, in June 1843. It can scarcely be necessary to say, that "an account of the disease which prevailed in the Penitentiary in the year 1823," was published by Dr. Latham.

during the same period of eighteen years. Now, if all these cases of invaliding were reckoned as deaths, the rate of mortality would be raised to rather more than fifty-eight per thousand (58·405), or 5 840 per cent. annually. This, however, would be a highly exaggerated estimate; for though it is true that many of the prisoners, thus invalided, have died soon after their release from prison, it is equally certain that the majority of them have recovered, and that in a large proportion of the cases the result would not have been fatal during the criminal's imprisonment, even had they been detained until the expiration of their sentences.

After a careful examination of the list of diseases which formed the grounds of pardon, and a consideration of all the facts bearing on this difficulty, I have formed the opinion that out of the 355 cases in which prisoners were invalided, 123 would in all probability have terminated fatally before the completion of the full terms of imprisonment, had no pardons been granted, and consequently that those 123 cases may be added to the number of deaths. According to this calculation, the annual mortality in the Penitentiary will have been 34·209 per thousand, or 3·420 per cent., as is shown in the subjoined Table.

Average number of prisoners.	Total number of Deaths in the Institution during 17 years.	Number of deaths annually per 1,000 prisoners.	Total number of pardons on medical grounds.	Probable total mortality found by adding 123 of the pardons to the deaths.	Annual ratio of probable mortality per 1,000 prisoners.
532	205	21·380	355	328	34·209

On referring to the 5th report of the Registrar-General, (page 35,) we find that this rate of mortality is more than twice as high as that to which all persons in the metropolis, of the same period of life as prisoners, were subject during the year 1841, and it would consequently seem that the criminal's liability to die was more than doubled by imprisonment in the Penitentiary.

Mortality per thousand of the inhabitants of the metropolis, between the ages of 15 and 70, during the year 1841	15,390.*

Two objections, however, may be urged against the fairness of this comparison. The first which I shall notice, is that the year 1841 was a healthy year, while during two of the 18 years over which the mortality tables of the Penitentiary extend, Asiatic cholera was prevalent, and caused 31 deaths amongst the prisoners. These deaths from cholera may certainly, with perfect fairness, be excluded

* The rate of mortality in 1841 is here taken as the standard for the annual mortality in the metropolis; because the census having been taken in that year, we know the *actual* population at different ages,—one of the elements in the calculation, by which we deduce the ratio of the deaths,—while, in other years, we are obliged to calculate from the *estimated* population. In subsequent parts of this paper, when the mortality from different diseases comes under discussion, other periods are referred to, viz., 1840 and 1841, taken together, and the year 1842. The reason of this is, that the Registrar-General's Reports give no account of the mortality of persons of different ages, from the various causes of death, in the year 1841 alone.

from the calculation in estimating the mortality of the Penitentiary; but even then, we find the ratio of deaths to the number of the living in the Penitentiary, and in the metropolis generally, to have been respectively 30·976 and 15·390 per thousand annually.

	Total mortality during the 18 years.	Annual rate of mortality per 1,000 Prisoners.
Mortality amongst the Prisoners in the Millbank Penitentiary during the years 1825 to 1842, exclusive of deaths from Asiatic cholera	174	18·147
Do. Do. with the addition of 123 pardons, which are to be reckoned as deaths . .	297	30·976

With this correction, therefore, the mortality in the Penitentiary still appears to have been excessive. But it may next be urged, that this excessive mortality should in great part be ascribed to the unhealthiness of the class of society from which the prisoners are chiefly derived. This second objection to the comparison drawn above, between the mortality in the Penitentiary and that amongst the free population of the metropolis, will be more conveniently considered, after we have inquired into the facts displayed in the statistical records of other penal establishments, and have determined how far a very high rate of mortality is to be regarded as their general characteristic.

The penal establishments respecting which I have been enabled to obtain the requisite data for calculating the proportion of deaths to the number of

prisoners, are the Houses of Correction and Gaols of the different Counties of England and Wales,* the Hulks of England,† the Maisons de Correction

* The statistics of the County Prisons of England and Wales have been extracted with much labour from the annual reports of the Inspectors of Prisons. The returns of the average daily number of prisoners given in those reports, include debtors as well as criminals in the cases of those County Gaols which receive both classes of prisoners. To ascertain the average number of criminal prisoners, I have deducted from the average daily number of all prisoners, the number of debtors at the date of the returns. The numbers thus obtained must approximate very closely to the real average numbers of criminal prisoners in the different gaols. In order however to avoid the imputation of inaccuracy, I have arranged in separate tables, those prisons which contained only criminal prisoners, or at least but very few debtors (see Table II. in the Appendix), and those in which the debtors are in considerable number (Table III.). The deaths of debtors are seldom noticed in the returns, but when they are mentioned I have subtracted them from the total number of deaths. Of the statistics of Lancaster Castle I have accurate accounts for a long series of years (see Table XXIV.); Captain Hansbrow, the Governor of that prison, having kindly furnished me with a return of the average number of the criminal prisoners, and the mortality in each year, from 1825 to 1843. The statistics of the Cold Bath Fields House of Correction and the Tothilfields Bridewell (Table IV.) were obtained from the Home Office in 1840. The details of the statistics of the Wakefield, Knutsford, and Devizes Houses of Correction, collected from the Reports of the Inspectors, will be found in Tables XXII. to XXV.

† The number of convicts confined in the Hulks, and the mortality they have suffered during 15 years, is shown in Table V., which has been formed from returns obtained from the Home Office.

et de Force, and the Bagnes or Hulks of France,* the Geneva and Lausanne Penitentiaries in Switzerland,† and several of the principal Penitentiaries or State Prisons of the United States.‡ The main re-

* A work compiled from official documents, by M. Raoul Chassinat, entitled, "Études sur la mortalité dans les Bagnes et dans les Maisons Centrales de force et de correction," and very recently published by authority of the Minister of the Interior, has furnished the data for estimating the mortality of criminals in the penal establishments of France.

† The statistics of the Penitentiaries of Geneva and Lausanne (see Tables VI. VII. XXIII. and XXIV. in the Appendix) have been gathered from the following sources: 1st, an article entitled Hygiène de la Prison Pénitentiaire de Genève," by M. Ch. Coindet, in the 19th volume of the Annales d'Hygiène Publique; —2nd, the "Examen Médicale et Philosophique du Système Pénitentiaire," of M. Gosse, published at Geneva, 1838; 3rd, the article on the "Régime Pénitentiare," by M. Moreau Christophe, in the 22nd volume of the Ann. d'Hygiène Publique; and 4th, two articles on the "Schweizer Strafanstalten," by M. Varrentrap, in the 1st and 2nd vol. of the Jahrbücher der Gefängnisskunde. Frankfort, 1842 and 1843.

‡ I have been enabled, through the kindness of W. Crawford, Esq., Inspector of Prisons, to collect very accurate information respecting the vital statistics of the American Penitentiaries, that gentleman having allowed me to consult his very extensive collection of statistical and other works relating to the state of prison discipline in America, and other countries. The data employed for the construction of the Tables VIII. to XIV. and XXVI. to XXXII. are principally derived from the following sources: the series of annual reports of the Inspectors of the Eastern Penitentiary of Pennsylvania; many of the annual reports of the Inspectors of the Penitentiaries or State Prisons of Auburn, Sing Sing, (or Mount Pleasant,) Charlestown, Wethersfield, and Maryland; Mr.

sults which these data have afforded, are shown in the annexed Table. Deaths from Asiatic cholera have been excluded from the calculation, in all cases except those of the French Hulks and Prisons. I have not been able to ascertain what amount of mortality was caused by the epidemic of cholera in the penal establishments of France.

Crawford's report on the Penitentiaries of the United States, ordered by Parliament to be printed, March 1835; Rapports sur les Pénitenciers des États-Unis par MM. Demetz et Blouet, Paris 1837; the annual reports of the Boston Prison Discipline Society; and the work of Dr. Julius, "Nord-Amerikas Sittliche Zustände," published at Leipzig in 1839.

Names of Prisons, &c.	Periods to which the data belong.	Average number of prisoners.	Total number of deaths during the whole period.	Annual ratio per 1,000 prisoners.
The 36 largest County Gaols and Houses of Correction in England.	5 years, 1838 to 1841	8,657	823	19·013
Wakefield House of Correction.	9 years, 1835 to 1843	561	135	26·732
Knutsford House of Correction.	7 years, 1834 to 1840	246	34	19·710
Lancaster County Gaol.	19 years, 1825 to 1843	263·736	102	20·355
Devizes House of Correction.	12 years, 1829 to 1840	174·666	32	15·767
Millbank Penitentiary.	18 years, 1825 to 1842	532	174*	18·147
The Hulks of England.	15 years, 1825 to 1831	3,389	1,980	38·938
The 19 Maisons centrales de Force et de Correction of France.	16 years, 1822 to 1837	M. 15,583 F. 4,768	M. 13,859 F. 3,030	M. 55·5 F. 39·5
Poissy (Maison centrale).	16 years, 1822 to 1837	852	417	30·5
Nismes (Maison centrale).	16 years, 1822 to 1837	1,373	1,345	61·3
Eysses (Maison centrale).	16 years, 1822 to 1837	1,423	1,981	86·9
The 3 Hulk Establishments or Bagnes of France.	16 years, 1822 to 1837	6,915	4,508	40·7
Brest (Bagnes).	16 years, 1822 to 1837	1,800	885	30·7
Rochfort (Bagnes).	16 years, 1822 to 1837	991	848	53·4
Toulon (Bagnes).	16 years, 1822 to 1837	4,182	2,775	41·4
The Geneva Penitentiary.	16 years, 1826 to 1841	56·911	24	26·380
The Lausanne Penitentiary.	15 years and 3 months, Oct. 1826 to Jan. 1841	88·144	52	38·690
The Maryland Penitentiary.	8 years, 1834 to 1840 & 1843	372	155	35·763
The Eastern Penitentiary of Pennsylvania.	14 years, 1830 to 1843	266·7	147	39·336
The Sing Sing State Prison of New York.	13 years, 1828 to 1840	763·384	300	30·822
The Wethersfield State Prison, Connecticut.	12 years, 1830 to 1837 & 1839 & 1842 to 1844	195	68	27·048
The Auburn Penitentiary, New York.	16 years, 1825 to 1840	613·62	186	19·019
The Charlestown State Prison, Massachusets.	12 years, 1829 to 1840	283	65	19·408

* Exclusive of prisoners pardoned on account of impaired health.

It will be seen that the annual rate of mortality ranges in the different prisons in England from 15·767 per thousand to 38·938 per thousand; in the prisons of the United States, from 19·019 to 39·336 per thousand; and in Switzerland from 26·380 to 38·690 per thousand; while in France the rate of mortality of the Hulks varies from 30·7 to 53·4 per thousand, and that of the Houses of Correction from 30·5 to 86·9 per thousand.

The annual rate of mortality amongst persons at liberty in the different counties or cities in which these prisons are situated is shown by the subjoined Table:

County or City.	Period.	Ages.	Sexes.	Ratio of deaths per 1,000 persons living.
England* (the Metropolis).	1841	15 to 70	Both sexes.	15·390
France† (the whole country).	1817 to 1831	15 to 70	Males. Females.	14·140 14·679
„ (the most healthy districts).	— —	— —	Males. Females.	13·178 14·510
„ (the most unhealthy districts).	— —	— —	Males. Females.	16·126 14·834
Paris‡	1817	15 to 70	Both sexes.	17·988
Switzerland§ (Geneva)	1814 to 1833	15 to 70	Both sexes.	16·165
United States‖ (City and County of New York).	Mean of 1838 & 1842	10 & upwards	Both sexes.	15·760

* Fifth Report of the Registrar-General, pp. 34, 35.

† Mém. sur les lois de la population, et de la mortalité en France, par M. Demonferrand, Journ. de l'École Polytechnique, Tome XVI. 26e cahier, Anné 1838, pp. 294, et seq.

‡ Recherches Statistiques sur la Ville de Paris, 1833, Tab. No. 7 and No. 20.

§ Recherches Historiques et Statistiques sur la Population de Genève, par M. Mallet, Ann. d'Hygiène, Tome XVII. p. 169.

‖ Census of the United States, 1840, and Annual Reports of

On a comparison of this Table with the preceding one, it will be seen that in all these countries the mortality of prisoners has much exceeded that of the free population. But it will be observed that the excess of mortality has been far greater in some prisons than in others. To what causes must we ascribe this difference in the rates of mortality presented by different penal establishments? This is the next question which offers itself for solution.

At first sight it might be supposed that the relatively low rate of mortality of some prisons and the high mortality of others, are measures of the healthiness or unhealthiness of these institutions, and of the different degrees in which the health of the prisoners is injuriously affected by the discipline, diet, and general arrangements to which they are subjected. A more exact inquiry, however, shows that such a supposition would be most erroneous, for we find that there are many circumstances independent of all systems of prison discipline and internal prison arrangements, which nevertheless greatly affect the number of deaths occurring in

the Interments in the City and County of New York. The rate of mortality is calculated from the population in 1840, and the mean number of the deaths in 1838 and 1842. The ages of the Black population being grouped differently in the Census and in the Annual Reports of the Interments in the City of New York, it is not possible to give the rate of mortality between the ages of 10 and 70. Hence when the mortality of the Black population alone, or of the whole population (Whites and Blacks together), is given in this paper, it has been necessary to include all ages above 10 years.

penal establishments. It is necessary that I should point out and illustrate the operation of the more important of these circumstances, before I attempt to define the real influence of imprisonment on the health and chances of life.

II.—Causes influencing the Rate of Mortality in Prisons.

The circumstances whose influence it is most necessary to consider before reasoning upon the data afforded by the mortality tables of a prison, are, first, the extent to which the practice is carried, of granting pardons to convicts in an impaired state of health; secondly, the degree of predisposition of the class of persons forming the population of the prison, to fatal disease; thirdly, the length of imprisonment which the convicts undergo; and, fourthly, their liability to endemic and epidemic diseases, from the situation of the prison.

I. *Pardons on Medical grounds.*—The necessity of inquiring whether the prisoners are invalided, or pardoned on account of impaired health, and to what extent this is practised, is clearly enough demonstrated in the case of the Millbank Penitentiary.

The table of deaths which actually occurred within that institution gives a rate of mortality of scarcely more than eighteen per thousand annually: but the prisoners pardoned on account of impaired health were very numerous,—so numerous, indeed, that, according to a fair estimate of the probable fatality of the diseases forming the grounds of pardon,

the rate of mortality, exclusive of the deaths from cholera, should be regarded as nearly thirty-one per thousand annually.

In no other penal establishment, probably, has the pardoning of prisoners on account of ill health been practised to so great an extent as in the Millbank Penitentiary; but undoubtedly it has not been confined to that institution. Prisoners in many of the county gaols and houses of correction of England have frequently received their pardons, on a representation of the impaired state of their health being made by the magistrates to the Secretary of State. The mortality really appertaining to the state of imprisonment in these establishments is therefore greater than is shown in the table at page 126.

Some data given in the Eighth Report of the Inspector of Prisons in the Northern and Eastern District, enable us to estimate the extent to which the number of deaths in the county prisons has been reduced by the pardoning of criminals in ill health. It appears that in the Wakefield, Liverpool, Preston, Lancaster, Chester, and Derby gaols, the number of prisoners pardoned on account of their bad state of health, during the years of which the statistics are given in the report above mentioned, has been 35, while the number of deaths has been 61.

Now, assuming that the prisoners who would have died had they not been released, bore the same proportion to the whole number pardoned as we have estimated them to have borne in the Millbank Penitentiary, we must regard the mortality of

these prisons, during the years to which the data belong, as 73·11 instead of 61. And if we further suppose that the number of deaths has been diminished, by the pardoning of criminals in ill health, to the same extent in the thirty-six principal gaols and houses of correction of England, as in the six prisons just named, we must estimate the real total mortality of the whole thirty-six prisons, during the five years, (1838 to 1842,) at 986·385 instead of 823, and their average annual mortality at 22·788 instead of 19·013 per thousand. This estimate is probably not far from the truth.*

Again, the comparatively low rate of mortality of some of the American prisons cannot be wholly

* The calculation by which this result is obtained is as follows:—The number of deaths during the last two or three years, in the six prisons named in the preceding page, has been 61, and the number of pardons 35. In the Millbank Penitentiary we reckoned 123 out of 355 pardons, or 346 per thousand, as deaths. Dividing 35, then, by $\frac{346}{1000}$, we find the number of pardons (namely, 12·110), which are to be reckoned as deaths, and to be added to the mortality of the six county prisons, and in this way we find 73·11 to be the probable total mortality of those prisons during the years in which 61 prisoners died in the prisons and 35 were pardoned. To find the probable mortality of the thirty-six county prisons during the last five years, we add to the deaths which occurred in the prisons, namely, 823, a number which bears the same proportion to 823 as 12·110 does to 61. This required number is 163·385, which being added to 823, makes the probable total mortality 986·385. The average annual mortality amongst 1,000 prisoners is of course ascertained by multiplying this number by 1,000, and dividing by the aggregate of the average numbers of prisoners in the several years, which was 43,285.

explained, except on the supposition that pardons have sometimes been granted to prisoners whose health has been failing, and who would have died if they had been kept longer in prison. No account of these pardons on medical grounds is given in any of the reports of the American Penitentiaries; yet that such pardons are granted is certain, from casual expressions used in some of their reports, and in pamphlets on prison discipline published in America.*

The number of prisoners pardoned under these circumstances, seems however to have been much less in the American prisons than at the Millbank Penitentiary. For in the Eastern Penitentiary during the first seven years after its establishment, only three prisoners appear to have been pardoned in a bad state of health.†

On the other hand, the comparatively high rate

* Two letters "On the abuse of the pardoning power" were published at Philadelphia in 1839; and in one of them the writer, Mr. Samuel R. Wood, for many years Warden of the Eastern Penitentiary, after condemning the facility with which pardons are granted to prisoners in America by the Executive, says, that one of the grounds often taken in the petition for pardon, is, "that he (the prisoner) is in delicate health, and that unless liberated he may die in prison." Again, the physician of the New Jersey Penitentiary, in his report for the year 1839, says, "There are generally from 10 to 20 on the sick list. Some of them are predisposed to consumption, and two or three have the disease in its confirmed state, and *will die in prison unless they are pardoned out.*"

† See the table of prisoners admitted into and discharged from the Eastern Penitentiary, at pages 98 to 119 of the Report of MM. Demetz and Blouet.

of mortality of the Hulks in England, 38·938 per thousand, is undoubtedly in part owing to the circumstance, that, as a general rule, convicts in the Hulks have not been pardoned on account of loss of health. The same has probably been the case in the French hulks and prisons, although other causes must have contributed to raise the mortality of those establishments to the high rate which they present.

2. *Differences of the Prisoners in respect of race, class, and consequent predisposition to disease.*— The extent to which the practice of invaliding or pardoning prisoners in bad health is carried, influences the *apparent* mortality in prisons, but there are other circumstances which cause the *actual* mortality to vary, quite independently of any effect which the discipline and internal arrangements of these establishments may produce upon the health. One of these circumstances is a difference in the race or class of persons constituting the population of the prisons, in regard to their state of health and liability to disease.

The influence which the condition of life of prisoners exerts on their rate of mortality while in confinement, was pointed out long since by M. Villermé.* He showed that the mortality in the different prisons in the Department of the Seine increased in a direct ratio with the wretchedness and poverty of the class of prisoners confined in them. The mean annual mortality of these prisons

* Annal. d'Hygiène Publique, tom. i. p. 3.

during the years 1815 to 1818 inclusive, was as follows:—

Prisons for persons in good or tolerable circumstances.	Grande Force . . .	1 in 40·88
	Madelonnette . . .	1 in 38·03
For persons of a poorer class.	Conciergerie . . .	1 in 32·06
	Petite Force . . .	1 in 26·63
	Sainte Pelagie . .	1 in 24·48
For persons who were nearly destitute.	Bicêtre	1 in 18·75
	Saint Lazare . . .	1 in 17·92
For beggars and vagrants who were totally destitute, and many diseased, aged, and infirm.	Depôt de Mendicité à Saint Denis	1 in 3·97

Other causes besides the mere poverty of the class whence the prisoners were derived, certainly contributed to increase the mortality of some of these prisons. And the comparatively low rate of mortality of those in which prisoners of a less destitute grade of society were confined, may be in part referred to the circumstance that such prisoners, having funds at their command, could obtain extra food and other comforts, even while in confinement. But still we cannot doubt that the more sound state of their constitutions had a large share in enabling the latter class of prisoners to resist the injurious influence of imprisonment, and that the intensity with which the same injurious influence operated on prisoners of a lower grade, was in a great measure owing to their having been rendered prone to disease by previous destitution and debauchery.

The importance of taking into account the different degrees of predisposition to disease which distinguish different classes of prisoners, before we draw inferences from the different rates of mortality of different prisons, is remarkably apparent in the case of the American Penitentiaries. We have already seen that the principal Penitentiaries or State Prisons of the United States vary considerably in the rate of mortality of the criminals confined in them. The annual proportion of deaths to the daily average of prisoners being in

The Sing Sing State Prison	30·822	per thousand.
The Eastern Penitentiary	39·336	,, ,,
The Maryland Penitentiary	35·763	,, ,,
The Wethersfield State Prison	27·048	,, ,,
The Charlestown State Prison	19·408	,, ,,
The Auburn State Prison	19·019	,, ,,

The high rate of mortality of the State Prison of Sing Sing is, as I shall presently show, in part due to its unhealthy situation. But this is not the case with the Eastern and Maryland Penitentiaries, both of which have elevated healthy sites. How then are we to account for the deaths being proportionally so much more numerous in these two Penitentiaries than in the State Prisons of Auburn, Charlestown, and Wethersfield? Must we infer that the relatively high rates of mortality of the two former institutions are wholly or chiefly due to an injurious influence arising from their construction, their internal ar-

rangements or system of discipline? If we were inclined to adopt such an opinion with regard to the Eastern Penitentiary, which is distinguished from the other prisons by a different construction and a different system of discipline, we should at once hesitate to do so, on remembering the high rate of mortality of the Maryland Penitentiary; for this institution is regulated after the very same system as the Auburn and Charlestown prisons, in which the rate of mortality is so low. A little further inquiry reveals to us another cause which will explain a great part of the excessive mortality both in the Eastern and in the Maryland Penitentiary. This cause is the peculiar predisposition to disease of a large proportion of the criminals confined within their walls. The number of prisoners in the Eastern Penitentiary during 14 years (1830 to 1843 inclusive) was on the average 267. Of this number, 165 were of the white race, and 102 of the dark races, chiefly negroes. In the Penitentiary of Maryland (a slave state) the proportion of prisoners of colour was even greater; the total number of prisoners received during eleven years being 1238, and 677, or more than half of that number, being people of colour. The presence of so large a proportion of individuals belonging to the dark races, who in a climate much colder than that of their native country, and in circumstances wholly unnatural to them, are known to suffer a very great mortality, would readily be admitted as a sufficient reason for the rate of mortality

of these Penitentiaries being very high. But the influence of this circumstance is even greater than could be anticipated. The subjoined Table gives some data for estimating the relative mortality of the white and coloured races amongst the free population of the cities and counties of Philadelphia and New York.

	Period.	Races.	Ages.	Population living.	Deaths.	Mortality per 1,000 living.
City and County of Philadelphia	1821 to 1830*	White	All ages	—	—	24·22
		Coloured	All ages			47·52
City and County of New York.	1840†	White	All ages	296,352	7,397	24·963
		Coloured	All ages	16,358	471	28·793
	Population of 1840, mean no. of deaths during 1838 and 1842†	White	10 and upwards	219,276	3,312	15·104
		Coloured	10 and upwards	13,076	350	26·766
	Ditto†	White	10 to 60	211,580	2,734	12·92
		Coloured	10 to 55	12,187	291	23·87
	Population and deaths, 1842†	White	10 to 60	220,991	2,952	13·35
		Coloured	10 to 55	12,736	297	23·32
		Whites, Males	10 to 70	107,941	1,626	15·063
		Females	10 to 70	118,502	1,618	13·653

In all the instances here cited, on whatever basis the calculation is made, as to place, year or age, the

* From Emerson's Medical Statistics.

† From the Annual Reports of the Interments in the City and County of New York. The population in 1840 is taken from the Census of the United States. For the opportunity of consulting these and several other statistical works I am indebted to W. Farr, Esq., of the General Register Office.

mortality is much greater amongst the coloured than amongst the white population; and it will be seen, that where the population under the age of ten years is excluded from consideration, the ratio of deaths amongst the coloured is nearly twice as high as amongst the white inhabitants. This difference in the mortality of the two races becomes much exaggerated in the state of imprisonment. The mortality of the white races is raised from 13 or 15 per thousand annually, to 20 per thousand; but that of the coloured race, which in the free condition ranges from 23 to 27 per thousand, has been during the last fourteen years as high as 70 per thousand in the Eastern Penitentiary, and even higher in the Wethersfield State Prison, during the last three years.

	Period.	Races.	Ages.	Population living.	Average annual No. of deaths.	Mortality per 1,000 living.
City and County of New York.	Population of 1840	White	10 and upwards	219,276	3,312	15·104
	Mean No. of deaths in 1838 & 1842	Coloured	10 and upwards	13,076	350	26·766
Eastern Penitentiary of Pennsylvania.	14 years, 1830 to 1843 inclusive	White	—	165	3·527	20·34
		Coloured	—	102	7·142	70·10
Wethersfield State Prison.	3 years, 1842 to 1844 inclusive	White	—	153·33	4·33	28·260
		Coloured	—	49·66	5	100·671

It is certain, then, that the coloured population of the United States suffers an appalling mortality in the state of imprisonment, and it follows that the number of deaths in the Penitentiaries and State

Prisons will, *cæteris paribus*, be greater in a direct ratio with the proportion of the coloured race amongst the prisoners. And when we compare the proportional numbers of the coloured and white races in the different Penitentiaries and State Prisons of the United States, it becomes apparent that the fact of the rate of mortality having been so much higher in the Eastern Penitentiary, in the Maryland Penitentiary, and in the Sing Sing State Prison, than in the State Prisons of Charlestown and Auburn, may in great part be referred to this cause.

Name of Penitentiary or Prison.	Number of Prisoners.				Proportions per cent. of coloured to total number.	Rate of mortality per 1,000 prisoners.
		White prisoners.	Coloured prisoners.	Total number of prisoners.		
Baltimore Penitentiary, Maryland	Received during 11 years, 1830 to 1840	561	677	1,238	54·685	35·763
Eastern Penitentiary, Philadelphia.	Average daily number during 14 years, 1830 to 1843	165·04	101·88	266·92	38·169	39·336
Wethersfield State Prison, Connecticut	Number on 31st March, 11 years, 1830, 1832, 1834 to 1839, and 1842 to 1844	148·454	45·727	194·181	23·646	27·048
Sing Sing State Prison, New York	Mean of the data in Table XIV. in the Appendix.	—	—	—	20·	30·822
Auburn State Prison, New York	Received in 15 years ending Dec. 1839	2,499	313	2,812	11·130	19·019
Charlestown State Prison, Massachusetts	Number at Michaelmas during 12 years, 1829 to 1840	247·33	31·75	279·08	11·379	19·408

In the Sing Sing State Prison, one fifth, in the Eastern Penitentiary nearly two fifths, and in the Maryland Penitentiary more than one half of the whole body of prisoners were negroes, mulattoes or Indians; while in the Auburn and Charlestown State Prisons, not one in nine belonged to the coloured races.

In the Wethersfield State Prison, the proportion of coloured prisoners was twice as great as in the Auburn State Prison, but much smaller than in the Eastern Penitentiary of Pennsylvania, and its rate of mortality was in like manner much higher than that of the Auburn Prison, but lower than that of the Eastern Penitentiary.

The foregoing facts prove incontestibly, that the diversity in the rates of mortality in the different Penitentiaries of the United States is in great part, though not wholly, due to the various proportions borne by the people of colour to the whole number of prisoners; and that any estimate of the relative salubrity of these establishments, and of the influence of their different systems of prison discipline on the health, which is formed without this circumstance being taken into account, must be incorrect and unjust.

The fact that bodies of prisoners differ greatly in their predisposition to disease, and in the amount of disease actually existing amongst them, deserves some consideration also, when we compare with each other the several penal establishments of England; for the high rate of mortality of the Hulks in England certainly depends in a great measure on

there being a large proportion of unhealthy and aged prisoners amongst the convicts in that establishment.

Until the recent change in the constitution of the Millbank Prison, the Hulks were the general depôt for all convicts sentenced to transportation. From this depôt the surgeons of the convict ships took the healthy and able bodied convicts, rejecting all such as they considered to be, from disease or infirmity, unfit for transportation to a penal settlement. A large accumulation of these rejected convicts consequently took place, and the mortality amongst them necessarily rendered the proportion of deaths amongst the whole body of convicts in the Hulks considerably higher than it would have been under other circumstances.

3. *Length of the terms of confinement.*—Another circumstance necessary to be taken into account previous to forming any opinion as to the causes on which the mortality of a prison depends, is the length of confinement which prisoners undergo. The average term of imprisonment varies greatly in different prisons, and, as a general rule, the rate of mortality is highest in those prisons where the terms of imprisonment are longest. This is apparent when we compare with each other the different prisons of England and the Continent.*

* The data from which the average duration of the terms of imprisonment in the different prisons has been calculated, are the total number of prisoners received into each prison, and the average number constantly in confinement during a series of years (see, for example, Table XV. in the Appendix).

Names of prisons.	Average duration of imprisonment.	Rate of mortality per 1,000 prisoners.
English County Gaols and Houses of Correction.	1 month 16 days	22·788
Hulks of England	10 months 12 days	38·938
Millbank Penitentiary	2 years	30·965
Geneva Penitentiary	1 year 8 months	26·380
Lausanne Penitentiary	1 year 4 months	38·690
Prisons of France	Men . . 3y. 1m. 6d. Women . 3y. 2m. 27d. Both Sexes, 3y. 1m. 17d.	55·5 39·5 53·6
Hulks of France	Men, 7 yrs. 10 mhs. 6 ds.	40·7

In the English county gaols and houses of correction, where the average duration of imprisonment is about six weeks, the annual rate of mortality, allowing for pardons on medical grounds, has been only 22·788 per thousand prisoners.

In the Geneva Penitentiary the average duration of imprisonment has been 20 months, and the annual rate of mortality 26·380 per thousand. In the Millbank Penitentiary the average term of imprisonment was rather more than two years, and the annual mortality was 30·965 per thousand; while in the French Hulks, where the mean length of imprisonment has exceeded seven years, the rate of mortality has been as high as 40·7 per thousand prisoners. To this general rule of the increase of the ratio of deaths in proportion as the duration of imprisonment is greater, there are however several exceptions even amongst the English and Continental prisons.

The Hulks of England, the Penitentiary of Lau-

sanne, and the French Maisons Centrales, all present higher rates of mortality, compared with other penal establishments in their respective countries, than can be explained by reference to the terms of imprisonment suffered by the criminals confined in them.

In the case of the English Hulks this is due to a cause which has already been mentioned, namely, the great accumulation of unhealthy convicts in those establishments.

The comparatively high rate of mortality in the Lausanne Penitentiary and French prisons is less easily accounted for.

The want of correspondence between the average terms of imprisonment and the rate of mortality is still more remarkable in the American Penitentiaries, especially when we compare them with the penal establishments of England and France.

Names of prisons.	Average duration of imprisonment.	Annual ratio of mortality per 1,000 prisoners.
Auburn State Prison	3 years, 3 months,	19·019
Maryland Penitentiary	3 years, 2 months, 12 days	35·763
Wethersfield State Prison	3 years, 1 month, 25 days	27·048
Sing Sing State Prison	3 years, 2 months, 2 days	30·882
Charlestown State Prison	2 years, 9 months, 4 days	19·408
Eastern Penitentiary	2 years, 1 month, 6 days	39·336

Thus amongst the American prisons the *lowest* rate of mortality is coincident (in the Auburn State Prison) with the *longest* average term of imprisonment, while the *highest* rate of mortality is

coincident (in the Eastern Penitentiary) with the *shortest* average period of imprisonment. And again, when we compare those American prisons in which the negroes are not very numerous, namely, the Auburn, Charlestown, and Wethersfield prisons, with the prisons of Europe, we find the latter presenting, for the most part, much higher rates of mortality, although the convicts confined in all of them, except the prisons and hulks of France, undergo shorter terms of imprisonment than do those in the American prisons.

These facts afford a fresh proof of the impossibility of explaining all the varieties in the rates of mortality of prisons, by reference to one cause. Other circumstances being the same, the rate of mortality will be low where the terms of imprisonment are comparatively short, and will be high where they are protracted; but the influence of length of imprisonment may be more than counterbalanced by other causes, tending to increase or diminish the proportion of deaths.

In consequence of the number of deaths in prisons being liable to increase or diminution from so many different causes, it is impossible to determine the exact influence of the duration of imprisonment, by comparing the rates of mortality in different penal establishments. But we attain this object more satisfactorily when we compare the rates of mortality to which prisoners are subject at different periods of their imprisonment in the same prison.

The medical and other records of the Millbank

Penitentiary have afforded me the requisite data for making such a comparison; and the results at which I have arrived are comprehended in the subjoined Table.*

Periods of Imprisonment.	First year.			Second year.		Third year.	Fourth year.	Fifth year.	Sixth year.	Total.
	First 3 months.	Second 3 Months.	Second 6 months.	Third 6 months.	Fourth 6 months.					
Number of prisoners exposed to the chances of disease and death during each period.	3571	3470	3264	2905	2472	1645	611	94	5	
		3365		2682						
Number of deaths at each period from all causes except Asiatic cholera and suicides.	1	15	22	36	25	41	10	—	—	150
		38		61						
Annual rate of mortality per 1,000 prisoners at each period of imprisonment.	1·120	17·291	13·477	24·44	20·226	24·924	16·202			
		11·292		22·744						

* The following are the data from which the number of prisoners exposed to the chances of disease and death, during the successive periods of imprisonment, has been computed.

In the course of the seventeen years, 1825 to 1841 inclusive, 3,615 prisoners were confined in the Penitentiary, under the penitentiary system. The convicts under sentence of transportation are not included. The imprisonment of 2,962 had already terminated previous to the 1st of January 1842, while 653 still remained in the institution at that date. Both the prisoners discharged and those that remained had reached very various periods of imprisonment. The periods are specified in the first line of the Table at the next page, and the number of the discharged prisoners, the number of those remaining, and the total number of both classes, who at the time of their release, or on the 1st of January 1842, had reached each of these periods of imprison-

Here we see that prisoners in the Millbank Penitentiary were liable to very different rates of mortality at different periods of their imprisonment;

ment, are given in the second, third, and fourth lines of this Table :—

Periods of imprisonment.	First 3 months.	Second 3 months.	Second 6 months.	Third 6 months.	Fourth 6 months.	Third year.	Fourth year.	Fifth year.	Sixth year.	Above 6 yrs.	Total.
Prisoners discharged within each period of imprisonment	50	74	218	221	251	1133	837	169	8	1	2962
Prisoners remaining, whose imprisonment had reached but not extended beyond each period.	38	40	80	198	196	74	25	2	—	—	653
Prisoners discharged and remaining, whose imprisonment had reached but not extended beyond each period.	88	114	298	219	447	1207	862	171	8	1	3615
Number of prisoners who entered on each period of imprisonment	3615	3527	3413	3115	2696	2249	1042	180	9	1	
Average number exposed to the liability of disease or death during each period.	3571	3470	3264	2905	2472	1645	611	94	5		

From these numbers we may calculate how many prisoners were exposed to the chance of disease and death during each period of imprisonment.

The whole 3,615 of course entered upon the first period of three months, but as the imprisonment of 88 did not extend beyond this period, there were only 3,527 who entered on the second period of three months; and again, as there were

the proportion of deaths to those living being, in the first year of imprisonment, 11·292 per thousand; in the second year, 22·744 per thousand; and in the third year, 24·924 per thousand; while in the fourth year it fell again to 16·202 per thousand. These numbers, however, by no means exhibit the whole influence of the duration of imprisonment on the production of fatal disease amongst the prisoners in the Millbank Penitentiary. It will be re-

114 whose imprisonment did not extend beyond the second period, only 3,413 entered on the third period, and thus the number of prisoners who entered on the successive periods gradually diminished, as is shown in the fifth line of the Table. Since, however, all the prisoners who entered upon each period of imprisonment did not remain during the whole period, these numbers are not exactly those we are seeking. They do not represent the number of prisoners exposed *throughout* each period to the chances of being diseased or dying. To obtain this, we must take the mean of the numbers at the commencement and at the end of each period. The reason for this is sufficiently obvious. For if we suppose the whole 3,615 to have entered the Penitentiary on the same day, and 88 to have been discharged at a nearly even rate during the first three months, leaving 3,527 at the commencement of the second three months, then the mean between 3,615 and 3,527, or 3,571, will be the mean average number in the institution during the first period of three months. In the same way, the mean between 3,527 and 3,413 may be assumed to be the average number in the Penitentiary during the second three months of their imprisonment, and so on for the remaining periods. These numbers, which are given in the sixth line of the above Table, are the numbers with which we have, in the text, compared the deaths and pardons at each period, in order to determine the influence of the duration of imprisonment.

collected, that besides those who actually died in the prison, there were many prisoners whose health became so much impaired as to endanger life, and who on this account were pardoned.

Now an examination of the proportion of prisoners, thus invalided or pardoned on medical grounds, at different periods of imprisonment, shows a progressive and rapid increase up to the fifth year; the proportion during the first year being 5·940 per thousand prisoners; in the second year 37·211 per thousand; in the third year 79·021; in the fourth year 117·839; and in the fifth year 127·658 per thousand. See the subjoined Table.

Periods of Imprisonment.	First year.			Second year.		Third year.	Fourth year.	Fifth year.	Sixth year.	Total.
	First 3 months	Second 3 months.	Second 6 months.	Third 6 months.	Fourth 6 months.					
Number of prisoners exposed to the chances of disease and death during each period.	3571	3470	3264	2905	2472	1645	611	94	5	
		3365		2682						
Number of pardons at each period.	—	1	19	44	56	130	72	12	—	334*
		20		100						
Ratio of pardons per 1,000 prisoners at each period of imprisonment.	—	1·152	11·641	30·292	45·307	79·021	117·839	127·658		
		5·940		37·211						

* It is to be remarked that the data in this and the two preceding tables refer only to 17 years, 1825 to 1841, and only to the Penitentiary prisoners (exclusive of the female convicts under sentence of transportation). Hence the number of deaths and pardons is not the same as in other tables in this paper.

It would not however be fair to measure the influence of protracted imprisonment on the rate of mortality, either by the number of pardons at different periods of the punishment, or by the deaths and the pardons, referrible to the corresponding periods, combined. For it must be borne in mind, that little more than one third ($\frac{346}{1000}$) of these pardons were cases of disease which would have proved fatal had imprisonment been continued; and further, that as the remission of the remainder of a convict's sentence would be recommended, and granted with less hesitation, when he had already endured imprisonment for a considerable period, than at the beginning of his punishment, the proportion of cases which would really have proved fatal has most probably been smaller amongst the prisoners pardoned in the third, fourth, and fifth years, than amongst those liberated in the first two years of their imprisonment. From this last consideration we should be inclined to admit, that the increase in the number of pardons in the more advanced periods of imprisonment had been much less than is represented in the foregoing table; but, on the other hand, we must recollect that the pardons were granted on account of diseases which destroy life slowly, and, consequently, that if the prisoners had remained in confinement, their deaths would have occurred in many cases six, nine or even twelve months later than the period at which they were pardoned.

Assuming that these sources of error compensate for each other, we shall find the rate of mortality, for each period of imprisonment, by adding together

the deaths, and rather more than one third ($\frac{346}{1000}$) of the pardons appertaining to the corresponding periods. Thus:

	No. of prisoners exposed to the chance of death.	Total Number of pardons.	Number of pardons to be added to the deaths.	Deaths.	Total mortality.	Ratio of mortality per 1,000 prisoners.
First year .	3365	20	6·920	38	44·920	13·052
Second year	2682	100	34·600	61	95·600	35·645
Third year .	1645	130	44·980	41	85·980	52·267
Fourth year	611	72	24·912	10	34·912	57·139
Fifth year .	94	12	4·152	—	4·152	44·170

The rate of mortality amongst the prisoners in the Millbank Penitentiary, then, appears to have been 13·052 per thousand during the first year of punishment; 35·645 during the second year; 52·267 during the third year; 57·139 during the fourth year; and 44·170 during the fifth year; the rate of mortality rising rapidly from the first to the third year, and slowly in the fourth year; but falling again in the fifth year. The objection may be raised, that there is too much of mere supposition in the process by which we attain to this result. But even admitting this, as to details, the correctness of the result in the main cannot be doubted. The mortality suffered by convicts during the first year of their imprisonment has been comparatively slight; it has been more than doubled during the second year, and has continued to increase to the end of the third year. (See the table at page 145.)

It is worthy of remark, too, that in the course of seventeen years (1825 to 1841), not one convict was

pardoned, and only one died during the first three months passed in the Penitentiary; although 3,571 were exposed to the chances of dying. Now if the proportion of prisoners dying had remained as small as this throughout their imprisonment, or if no prisoners had been detained for more than three months, the annual rate of mortality would have been very little more than 1 (1·120) per thousand, and the Millbank Penitentiary would, as far as could have been inferred from the mortality occurring in it, have appeared to be by far the healthiest prison in England. This fact completely exposes the fallacy of the comparisons which are often drawn between the rates of mortality in different prisons, without reference to the length of the terms of imprisonment.

If any confirmation of this assertion were required, it would be found in the statistical facts relating to another class of prisoners who were confined in the Millbank Penitentiary, and to whom no reference has yet been made—I mean the military offenders who had been sentenced by court martial. Of this class of prisoners, 3,592 were confined in the Penitentiary between the 18th of November 1837 and the 31st of December 1842, and during the whole of that period three died, and four were pardoned on medical grounds, the annual proportion of deaths to the average daily number of soldiers in confinement being only 4·601 per thousand, of pardons 6·135 per thousand, and of deaths and pardons together only 10·736 per thousand.* This exceedingly low

* See Table XVI. in the Appendix.

ratio of deaths, and pardons on medical grounds, amongst the soldiers in the Penitentiary, was entirely owing to the short period during which the majority of them were confined, their average term of imprisonment being only two months and five days.

In the statistical account of the mortality in the prisons of France compiled by M. Raoul Chassinat, and recently published, one of the points which is fully illustrated by tables, is the influence of the duration of imprisonment on the mortality of convicts. The data comprised in the subjoined Table, which gives the rate of mortality of male convicts during each year of imprisonment in twelve great prisons of France, are borrowed from that work.

Names of prisons.*	Rate of mortality per 1,000 in different years of imprisonment.										
	1st year.	2nd year.	3rd year.	4th year	5th year.	6th year.	7th year.	8th year.	9th year.	10th year.	Above the 10th year.
Beaulieu .	39·8	56·9	40·0	66·3	49·0	37·0	34·7	39·2	15·1	16·1	00·0
Clairvaux .	29·8	61·0	69·4	58·1	67·3	152·4	165·5	213·9	131·4	135·4	98·9
Embrun .	46·2	63·1	45·0	45·6	19·8	25·4	33·6	8 5	12·3	00·0	34·4
Ensisheim .	41·4	60·4	55·9	57·7	46·1	22·6	22·7	36·2	29·1	5·4	44·4
Fontevrault .	29·5	43·3	46·8	45·7	57·8	50·7	66·1	72·4	67·7	45·0	82·0
Loos .	47·2	65·9	72·1	43·5	26·3	16·5	31·3	11·4	8·6	30·3	71·4
Melun .	28·5	45·7	47·8	30·6	22·5	23·8	18·0	19·3	13·0	14·0	35·0
Mount St. Michel	26·8	58·1	69·7	59·5	45·0	32·2	23·4	12·8	5·8	6·8	66·6
Nismes .	46·5	71·2	85·8	85·3	48·9	41·8	30·5	16·4	37·9	15·8	22·7
Poissy .	24·7	41·6	32·3	36·8	22·4	8·0	17·2	00·0	00·0	86·9	00·0
Riom .	48·5	76·6	94·7	79·8	71·5	39·6	13·6	24·7	27·6	62·5	55·5
Rennes .	39·4	44·0	50·7	55·6	17·9	44·0	33·0	9·9	25·3	13·1	54·0
Average .	37·35	57·48	59·18	55·37	41·20	41·17	40·80	38·72	31·15	35·94	47·07

* The Maisons Centrales of Eysses, Gaillon, and Limoges, *are omitted* in this table, because the mortality in those prisons *seems to have* been rendered excessive by [illegible] uses.

Here, as in the Millbank Penitentiary, the prisoners have suffered much less mortality during the first year of their imprisonment, than at subsequent periods, and less in the second than in the third year. In these French prisons, however, where the mean annual rate of mortality amongst the male convicts is as high as 47·07 per thousand, the difference between the first year's and second year's mortality, and between the second and the third year's, is not so great as it has been in the Millbank Penitentiary. The injurious influences to which the high rate of mortality is owing, have been felt even at an early period of imprisonment.

The Eastern Penitentiary of Philadelphia is the only American Prison, the published records of which furnish the means of ascertaining the proportional numbers of the deaths amongst the convicts at different periods of their imprisonment; and the data, in the case of this prison, only extend over the first seven years succeeding its establishment, when the number of convicts was small. Still the results they afford, confirm, to a certain extent, those already detailed. The mortality of the convicts during the first three months' imprisonment was only 11·851 per thousand; during the first year 22·071 per thousand; and during the second year 47·729 per thousand; during the third year it was only 38·647; and in the fourth year only 24·719 per thousand.*

* The data from which these results have been calculated, are contained in the Table given at the next page:—

Here then the mortality was comparatively low during the first year's imprisonment, and rose greatly in the second year. It is probable too that the difference between the first year's and the second year's rate of mortality would have been even greater, but for the unhealthy condition of many of the black convicts at the time of their re-

	First year.			Second year.		Third year.	Fourth year.	Fifth year.	Sixth year.	Seventh year.	Total.
	First 3 months.	Second 3 months.	Second 6 months.	Third 6 months.	Fourth 6 months.						
Discharged .	7	11	59	75	87	46	16	9	1	1	312
Remaining .	37	34	68	89	82	43	13	4	6	9	385
Discharged and remaining	44	45	127	164	169	89	29	13	7	10	697
Prisoners who entered on each period of imprisonment.	697	653	608	481	317	148	59	30	17	10	
Exposed to disease and death throughout each period.	675	630·5	544·5	399	232·5	103·5	44·5	23·5	13·5	5	
	589			314·5							
Deaths . . .	2	5	8	10	3	3	1	—	—	1	33
Pardons . .	—	—	—	—	2	1	—	—	—	—	3
Rate of mortality per 1,000, the pardons being reckoned as deaths.	11·851	31·720	29·559	50·125	43·017	38·647	24·719	—	—	200·000	
	22·071			47·729							

This table shows, in the first three lines, the periods of imprisonment which had been reached by the 312 prisoners who were discharged from the Eastern Penitentiary during the seven years, 1830-1836, and by the 385 who remained in the institution on the 31st December 1836. The rest of the table will be easily understood if it be compared with those given at pages 145 and 146, with the note at page 145.

ception, which rendered them incapable of bearing imprisonment, even for one year. The ratio of deaths fell during the third year of imprisonment, and again still more in the fourth year; but much importance cannot be ascribed to this result, since the number of prisoners exposed to the chance of dying at a period of imprisonment more advanced than the second year, was too small to afford a safe basis for statistical reasoning. I must however remark, that in the Millbank Penitentiary, and also in the prisons of France, the mortality has been greater amongst the prisoners who were undergoing their second, third or fourth year of imprisonment than amongst those who had been longer in confinement, so that it would seem as if prisoners who were of feeble constitution, or predisposed to disease, generally fell victims to the injurious influences attending imprisonment before the end of the fourth year of their confinement, while those who were able to support their punishment until that period, without serious deterioration of health, were proof against the causes of disease to which they were exposed.

4. *The Locality.*—The circumstances whose influence we have hitherto examined, namely, the number of prisoners pardoned on account of sickness, their original condition as regards predisposition to disease, and the length of their sentences, should always be first considered in drawing a comparison between the rates of mortality of several different prisons. If all the prisons be found to stand on a

nearly equal footing as regards these three circumstances, and the mortality of one of them be excessive, we shall then, and not till then, be justified in concluding that some cause unfavourable to health exists either in the locality of this prison, in its construction, in the diet, clothing and lodging of the prisoners, or in the discipline to which they are subjected. The influence of the locality should in this case be determined before the excess in the number of deaths is imputed to the internal arrangements of the establishment, and the only sure evidence of the mortality being increased by a noxious influence of the locality, will be the prominent part played by endemic diseases amongst the causes of death. Hence it will be most important to determine the relative number of deaths caused by diseases of this class in the different prisons, for we may by this means be enabled to refer to an influence of situation, the excessive mortality which would else be attributed to the discipline or other internal arrangements.

These remarks may be illustrated by the example of the American prisons. The rate of mortality, as we have seen, varies greatly in these prisons. In two of them, namely, the Auburn and Charlestown State Prisons, it is under 20 per thousand annually; while in three others, the Maryland Penitentiary, the Eastern Penitentiary, and the Sing Sing State Prison, it is upwards of 30 per thousand annually. The excessive mortality is entirely accounted for, in the case of the Maryland Penitentiary, by the num-

ber of coloured prisoners received into that institution. But in the Sing Sing State Prison, and in the Eastern Penitentiary, the proportion of coloured prisoners is not large enough to explain away more than a part of the excess in the ratio of deaths. It is not difficult to demonstrate this, and we may even calculate with tolerable accuracy, how much of the excess of mortality is accounted for, by the coloured population of these prisons, and how much remains to be explained by other causes.

In the Eastern Penitentiary the rate of mortality of the white prisoners has been about 20 per thousand annually, and that of the coloured population 70 per thousand annually. But in the Auburn and Charlestown Prisons, neither of these classes of prisoners can have suffered so high a rate of mortality, for the annual ratio of deaths amongst both the white and the coloured prisoners, taken together, has been under 20 per thousand. It is probable that more convicts have been pardoned in a state of disease, which would have terminated in death, in these prisons, than in the Eastern Penitentiary; but that is a question which we are not now considering;—we are looking merely to the mortality actually occurring in the different prisons; and, making our calculations on this basis, we must assume that the ratio of deaths amongst the coloured convicts in the Auburn Prison has not been more than 60 per thousand annually, nor the mortality amongst the white prisoners more than 16 per thousand annually. According to this estimate, which cannot

be far from the truth, we can account for the whole number of deaths in all the American prisons, except the Eastern Penitentiary, and the Sing Sing State Prison, but in these prisons some excess of mortality still remains to be explained.

The subjoined Table shows the rates of mortality (exclusive of deaths from cholera) which have actually prevailed in six of the American prisons; and also the rate which should appertain to each, according to the estimate of the mortality of white and coloured prisoners just given.

Names of prisons.	Auburn State Prison.	Charlestown State Prison.	Sing Sing State Prison.	Wethersfield State Prison.	Eastern Penitentiary, Pennsylvania.	Maryland Penitentiary.
Proportion of coloured prisoners to the whole body of prisoners.	11·130 per cent.	11·379 per cent.	20 per cent.	23·646 per cent.	38·169 per cent	54·685 per cent.
Actual rate of mortality per 1,000, exclusive of deaths from cholera.	19·019	19·408	30·822	27·048	39·336	35·763
Hypothetical rate of mortality, estimating that of the white prisoners at 16 and the coloured at 60 per 1,000.	20·897	21·006	24·800	26·404	32·973	40·061

This table shows that the actual rate of mortality is, in two of these prisons (those of Auburn and Charlestown), slightly below, and in one (the Maryland State Prison) considerably below, the rate theoretically assigned to them. In the Eastern Penitentiary, and in the Wethersfield and Sing Sing State Prisons, on the contrary, the actual rate of mortality presents an excess, which in the Wethers-

field prison is very trifling in amount, but in the two other prisons is equal to six deaths annually per thousand prisoners.

Let us now inquire into the nature of the diseases causing death in these different American prisons, and we shall, I think, find a satisfactory explanation of the excess of mortality in the prison of Sing Sing.

If Tables XXVI. to XXXII., in the Appendix to this paper, be examined, it will be seen that I have classed under distinct heads the following causes of death:—1. Fevers; including remittent and typhus or continued fever, but exclusive of the exanthemata. 2. Bowel complaints; comprehending common cholera, diarrhœa, dysentery, enteritis, and ulceration of the bowels; and 3. Hepatic diseases, comprehending hepatitis, diseased liver, and jaundice. Now with regard to the bowel complaints, it will, I think, be admitted, that where they are remarkably prevalent, in a severe form, through a series of years, they may safely be referred to an endemic cause, of the nature of malaria. That they have been thus prevalent in the Sing Sing State Prison is certain; for the mortality caused by them in this prison during the eleven years, 1830 to 1840 inclusive, has been 4·862 per thousand annually, while amongst the population of the city and county of New York above the age of 10 years, the mortality by the same diseases has been only ·884 per thousand annually.*

* In addition to the evidence drawn from the number of deaths from fevers and bowel complaints, which is adduced in the text, I

	Amongst the population above the age of 10 years in the City and County of New York during the year 1843.	Amongst the Prisoners in the Sing Sing State Prison.
Annual mortality per 1,000 by cholera, diarrhœa, dysentery, enteritis, and ulceration of the bowels.	·884*	4·860

With respect to the fever, more hesitation may at first be felt, in admitting that it should be classed with diseases arising from insalubrity of the locality. But, from the following considerations, it seems to me far more probable that, at least, the majority of the fatal cases of fever were due to this cause, than that they were cases of typhus generated by causes existing within the prison itself, or introduced from without by contagion or infection. In the first place, there is in the Sing Sing prison no crowding of prisoners in confined dormitories, such as produced or fostered the gaol fever of a former period: each convict occupies a separate cell at night, and during the day works either in a spacious workshop, or in the open air. In the second place, the introduction of contagious typhus is improbable, both from

may refer, in proof of the prevalence of these diseases in the Sing Sing prison, to a table of the predominant diseases amongst the hospital cases in that prison during five years, which is given at page 39 of Mr. Crawford's "Report on the Penitentiaries of the United States." It will there be seen that out of 3,357 cases of sickness, exclusive of Asiatic cholera, 236 were cases of dysentery, 707 cases of diarrhœa, 52 cases of common cholera, and 96 cases of fever.

* Gastritis is here included with enteritis.

the absence or rarity of intercourse between the prisoners and other persons who are at liberty, and also from the situation of the prison at a distance from any large town. Lastly, positive arguments in favour of the fever in Sing Sing State Prison being of malarious origin, are furnished in the concurrent prevalence of diarrhœa and dysentery; in the term "congestive fever," in many instances used by the physician—a term commonly applied by American writers to a fever of undoubted malarious nature; and, lastly, in the situation of the building. It stands on a low flat, at the foot of a hill, which completely overlooks it, and close to the bank of a very large tidal river, the Hudson.

The mortality from fevers, however, in this prison has not been so excessive as the mortality from bowel complaints. The average annual ratio of deaths from fever, in the Sing Sing Prison, has been 3·093 per thousand, and in the city and county of New York, 1·400 per thousand.

	Amongst the population above the age of 10 years, in the City and County of New York during 1842.	Amongst the Prisoners in Sing Sing State Prison.
Annual mortality per 1,000, from fevers, not including the exanthemata.	1·400	3·093

With the fevers and bowel complaints of the malarious districts of hot climates, hepatic diseases, it is well known, are generally, if not always, associated. When, therefore, in particular localities within the temperate zone, we find affections of the

liver, as well as fevers and bowel complaints, producing an unusual number of deaths, we are justified in inferring, if no other obvious cause be present, that the excess of mortality from these liver affections, like the increased number of deaths from fever and bowel complaints, has been caused by a local malarious influence; and we must, I think, come to this conclusion with respect to the hepatic affections which have caused several deaths in the Sing Sing Prison. The mortality from diseases of the liver in that establishment has been more than twice as great as amongst the population of the city and county of New York.

	Amongst the population above 10 years of age in the City and County of New York during 1842.	Amongst the Prisoners in Sing Sing State Prison.
Annual mortality per 1,000 from hepatic diseases.	·334	·773

Dropsy, too, has caused an extraordinary number of deaths in the Sing Sing Prison, and has, I have no doubt, been the effect of malarious influence. For on the coast of Africa, and in the West Indies, where bowel and liver complaints, and fevers, are rife, dropsies also are amongst the diseases most fatal to our troops. In order, however, to avoid the appearance of wishing to exaggerate the influence of the locality of Sing Sing, I shall not here class dropsies with the other endemic diseases.

We have now to compare the mortality in Sing Sing State Prison from fevers, bowel complaints, and hepatic affections, with the mortality from the

same diseases in the other American prisons. This comparison is made in the subjoined Table.

Names of Prisons.	Sing Sing State Prison.	Eastern Penitentiary.	Maryland Penitentiary.	Auburn State Prison.	Charlestown State Prison.
Annual mortality per 1,000 prisoners, from fevers.	3·093	1·070	0·000	1·099	1·961
Annual mortality per 1,000 prisoners, from bowel complaints.	4·860	·802	1·709	·610	·653
Annual mortality per 1,000 prisoners, from hepatic affections.	·773	·000	·569	·366	·000
Annual mortality per 1,000 prisoners, from fevers, bowel complaints, and hepatic affections.	8·726	1·872	2·279	2·075	2·614

The much greater number of deaths caused by these diseases in the Sing Sing Prison than in the other prisons, is at once apparent. In the Auburn and Charlestown State Prisons, and in the Maryland and Eastern Penitentiaries, the annual mortality from fevers, bowel complaints and liver affections, has been on the average 2·210 per thousand. In the State Prison of Sing Sing it has been 8·726 per thousand, so that there has been in that prison an excess in the annual mortality, by these classes of diseases, equal to 6·516 per thousand prisoners. We are now, therefore, in a position to explain the whole mortality in the Sing Sing Prison.

It will be remembered that the rate of mortality in this prison has been 30·822 per thousand annually, while the rate of mortality in the Auburn Prison, which is situated in the same State, and regulated

according to the same system of discipline, has been only 19·019 per thousand annually. The greater number of coloured prisoners in the former prison, accounted in part for its high rate of mortality, but, due allowance being made for this circumstance, the annual ratio of deaths should still have been only 24·800 per thousand prisoners; so that there remained an excess of rather more than six deaths annually per thousand prisoners. The cause of this we have now discovered in the prevalence of fever and endemic diseases of the digestive organs, which have given rise to a much greater mortality in the Sing Sing Prison, than in the other American prisons, the excess in the average annual proportion of deaths being more than six per thousand prisoners. Although, therefore, the difference is so great between the rate of mortality of Sing Sing and that of the Auburn prison, yet there is every reason to suppose that the internal prison arrangements do not produce a more injurious effect upon the health in one of those establishments than in the other, the excess in the number of deaths in the Sing Sing prison being entirely referrible to the number of the coloured prisoners, and to the insalubrity of the locality.

Leaving now the subject of the American Penitentiaries, let us inquire whether the influence of an unhealthy site has in an important degree affected the mortality of any of the *Prisons of England*. This inquiry is especially interesting in relation to the Millbank Penitentiary; but it has appeared to me, that both the interest and value of the conclu-

sion arrived at would be increased by subjecting the principal County Gaols and Houses of Correction, together with the Penitentiary, to a comparative examination. The decision of this question, in reference to any prison, necessarily depends on the number of deaths caused there by endemic disease. No other kind of proof, as I have before remarked, can determine satisfactorily, whether a malarious influence has raised the rate of mortality amongst the prisoners. Hence we must at present confine our attention to those prisons whose published statistics make us acquainted with the causes, as well as the number, of the deaths occurring within their walls. The Medical Records of the Millbank Penitentiary, from the period of the re-opening of the institution to the end of the year 1842, are in this respect as complete as could be wished. Very excellent tables of the deaths and causes of death in the Lancaster County Gaol, and the Wakefield, Knutsford and Devizes Houses of Correction, have also been furnished by the surgeons of those prisons to the Inspectors of the Northern and Eastern, and the Home Districts, who have published them in their Reports. The periods over which the data in these tables extend, are sufficiently long, and the deaths are sufficiently numerous, to form a tolerably sure basis for an opinion as to the prevalence of different classes of diseases in the prisons just named.

The accounts of the deaths and causes of death in the majority of the English County Prisons, which are contained in the Reports of the Inspectors, either do

not reach back more than two or three years, or have been published irregularly. They cannot, therefore, be admitted as trustworthy evidence of the kind of diseases which are usually prevalent in the individual prisons. Taken in the aggregate, however, these data are valuable, since they afford us the means of estimating the average mortality from different classes of diseases in the prisons of England generally, without reference to particular prisons. Such, then, are the data which we must examine, with the view of determining whether in the case of the English prisons, as in those of America, the influence of a malarious site has in particular instances produced a rate of mortality greatly surpassing the average rate in other similar establishments.

From the general Tables of deaths in the Millbank Penitentiary, in the Lancaster County Gaol, in the Wakefield, Devizes and Knutsford Houses of Correction, and in twenty-nine other English prisons, combined in two groups,* I have extracted all the deaths due to those diseases which most frequently arise from endemic causes.

I have arranged those diseases in three classes, namely,—1, Fevers; 2, Bowel Complaints; and 3, Hepatic Affections; and have calculated the annual rate of mortality caused in the different prisons and groups of prisons by these different classes of diseases. The results thus obtained, are shown in the following abstract Table:—

* See Tables XVIII. to XXV. in the Appendix.

Names of Prisons.	Periods to which the data belong.	Aggregate of the yearly averages of prisoners.	Total mortality.		Mortality by fevers.		Mortality caused by bowel complaints.		Mortality by hepatic affections.		Mortality by the three classes of diseases combined.	
			No.* of deaths	Annual No. per 1,000 prisoners.	No. of deaths	Annual No. per 1,000 prisoners.	No. of deaths	Annual No. per 1,000 prisoners.	No. of deaths	Annual No. per 1,000 prisoners.	No. of deaths	Annual No. per 1,000 prisoners.
Millbank Penitentiary.	18 years, 1825 to 1842.	9,588	174	18·147	35	3·650	18	1·877	1	·104	54	5·632
Wakefield House of Correction.	9 years, 1835 to 1843.	5,050	135	26·732	29	5·742	16	3·168	1	·198	46	9·108
Devizes House of Correction.	12 years, 1829 to 1840.	2,096	32	15·267	9	4·293	4	1·908	—	·000	13	6·202
Lancaster County Gaol.	19 years, 1825 to 1843.	5,011	102	20·355	5	·997	16	3·193	1	·199	22	4·390
Knutsford House of Correction.	7 years, 1834 to 1840.	1,725	34	19·710	3	1·739	3	1·739	—	·000	6	3·478
12 English County Prisons, chiefly Houses of Correction. See Table XX.†	Various between 1837 and 1842.	12,046	196	16·270	20	1·660	15	1·245	2	·166	37	3·071
17 English Prisons, chiefly County Gaols. See Table XXI.	Various periods. See Table XXI.	8,427	181	21·478	16	1·898	10	1·186	5	·593	31	3·678
All the County Gaols and Houses of Correction.	As stated above.	34,355	680	19·793	82	2·386	64	1·863	9	·261	155	4·511

The first point here elucidated, to which I would draw attention, is the comparative mortality by fevers, bowel complaints and hepatic diseases, in the Mill-

* Exclusive of those caused by Asiatic cholera.

† Table XX. in the Appendix includes these twelve prisons, and also the Wakefield, Devizes and Knutsford Houses of Correction, and the Lancaster County Gaol.

bank Penitentiary and in the English County Prisons respectively. The opinion generally entertained respecting the site of the Penitentiary is so unfavourable, that few persons would have been surprised had the mortality from diseases of the endemic class appeared far greater in that institution than in any other prison in England, and many perhaps will still be slow to believe what is here demonstrated by indisputable facts,—that the proportion of deaths caused by this class of diseases has in the principal county prisons, taken collectively, been only $\frac{1}{5}$th less, while in some of the prisons it has been considerably greater, than in the Penitentiary. The average annual mortality by all the diseases which can be ascribed to the influence of malaria has been 4·511 per thousand prisoners in the County Goals and Houses of Correction, and 5·632 per thousand in the Millbank Penitentiary.

This excess of mortality on the side of the Penitentiary has been almost entirely due to fever; the proportion of deaths caused by bowel complaints has been scarcely at all greater in that institution than in the county prisons.

Such is the result of a comparison of the Penitentiary with other English prisons taken collectively. When we examine the statistics of the individual county prisons, we find that in at least two instances they have suffered a greater mortality from fevers, bowel complaints and hepatic diseases, than the Penitentiary. For in the Wakefield House of Correction the mortality by fever alone has been 5·742

per thousand prisoners; by bowel complaints alone, 3·168 per thousand prisoners; and by all diseases which can be regarded as of endemic origin, taken together, 9·108 per thousand prisoners,—a rate of mortality which is $\frac{4}{5}$ths higher than has been experienced from the same diseases by the prisoners in the Penitentiary. In the Devizes House of Correction, also, the mortality by these diseases has been greater than in the Penitentiary, though the excess has not been so considerable as in the Wakefield House of Correction.

In the above comparison of the mortality from fevers, bowel complaints and liver affections, in the Penitentiary and other English prisons, account has been taken only of the deaths which have actually occurred in the institution. It seems unnecessary to make any allowance for the cases of prisoners pardoned while in ill health. For the diseases now under consideration are for the most part of an acute character, and when they become chronic, though often obstinate, are seldom incurable. The prisoners pardoned on account of these diseases must, therefore, in all prisons have been few, and their number has probably not been greater in the Millbank Penitentiary than in the County Gaols and Houses of Correction. But even if we were to add one-fourth of the pardons on account of these diseases, (·469 per thousand prisoners annually,) to the deaths in the case of the Penitentiary, and were to make no such addition in the case of the other prisons, the mortality by fever, bowel complaints

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and hepatic affections, in the Penitentiary, would still not be greater than in the House of Correction of Devizes, and would still be $\frac{1}{3}$rd less than in the Wakefield House of Correction.

There are other prisons, besides those of Wakefield and Devizes, in which diseases of the endemic class have produced a greater mortality than in the Penitentiary; but the published statistics do not in those instances extend over such long periods, and consequently would not form examples so trustworthy and decisive; and I abstain from referring to them individually. The evidence I have adduced is quite sufficient to establish the fact, that the site of the Penitentiary is not so pre-eminently insalubrious as has been supposed.

Another result of more general professional interest, and greater importance, may be deduced from this inquiry respecting the mortality caused by fevers, bowel complaints and hepatic diseases in the prisons of England. We have seen that the average proportion of deaths from these diseases, amongst the criminals confined in the principal county prisons, has been 4·511 per thousand annually. An examination of the reports of the Registrar-General, shows that the mortality from the same causes amongst persons in the metropolis, of the same period of life as prisoners, amounts to only 1·226 or 1·418 per thousand annually. The mortality from these diseases, therefore, during recent years has been more than three times as great in the county prisons of England, as amongst

the general population of London. This is shown in the subjoined Table. The mortality in the prisons, from the diseases referred to, is there compared with the mortality caused by the same diseases in the metropolis, amongst persons between the ages of 15 and 60, during the years 1840 and 1841,* and also amongst persons between the ages of 15 and 70 during 1842,† a year in which bowel complaints were unusually fatal.

	Periods.	Number of prisoners or population.	Total mortality.		Mortality by fever.		Mortality by bowel complaints.		Mortality by jaundice, hepatitis, and diseased liver.		Mortality by these three groups of diseases.	
			No. of deaths.	Annual number per 1,000 living.	No. of deaths	Annual number per 1,000 living.	No. of deaths	Annual number per 1,000 living.	No of deaths	Annual number per 1,000 living.	No. of deaths	Annual number per 1,000 living.
33 County gaols and houses of correction.	Various	34,355	680	19·793	82	2·386	64	1·863	9	·261	155	4·511
Population of the metropolis between the ages of 15 and 60.	2 years, 1840 and 1841.	1,182,402	30,518	12·994	1276	·546	910‡	·388	681	·292	2867	1·226
Population of the metropolis between the ages of 15 and 70.	1 year, 1842.	1,275,842	18,764	14·707	705	·552	687‡	·538	418	·327	1810	1·418

‡ Gastritis included.

* See the Fifth Report of the Registrar-General, p. 456 et seq.; where a table is given, showing the deaths from various causes at this period of life, in the two years 1840 and 1841, and the annual deaths to 1,000,000 living at each period of life.

† The deaths in the metropolis during the year 1842, from different diseases, and at different ages, are given at p. 296 to 303 of

In this comparison, hepatic diseases have been included with fevers and bowel complaints. This has been done, rather with the view of making the basis of the comparison, in the case of these English prisons, the same as in that of the American Penitentiaries, than from the belief that the affections of the liver, which have been most frequently fatal in the English prisons, have been due to the same causes as the bowel complaints and fevers. Hepatic affections, in fact, appear to have been less fatal in English prisons than in London, amongst persons of a corresponding period of life. It is from fevers and bowel complaints, that the excess of mortality just now pointed out has arisen. The annual average rate of mortality from fevers has been 2·386 per thousand in prisons, and only ·546 or ·552 per thousand in the metropolis. And the mortality from bowel complaints, which even in 1842 was only ·538 in the metropolis, and in the years 1840 and 1841 only ·388 per thousand, has been on the average 1·863 per thousand in the prisons. Fevers, therefore,

the Fifth Report of the Registrar-General. The population of the metropolis of the ages of 15 to 70 for the year 1842, has been calculated by multiplying the population, at the same period of life for 1841, by 1·0175,—the probable annual rate of increase of the population of the metropolis between the ages named, during the 10 years from 1831 to 1841. The actual annual rate of increase of the whole female population of the metropolis between 1831 and 1841 was 1·01543 (see the Registrar-General's Fifth Report, p. 448). But tables of the increase of the population of all England, given in the Fourth Report, show that the increase is greater in the middle than at the two extremes of life.

have been between four and five times as fatal, and bowel complaints at least four times as fatal, in prisons as in the metropolis.

Fevers and bowel complaints are far from being the diseases which produce the greatest mortality amongst prisoners. Fever, formerly such a scourge, is now comparatively a rare disease in the English gaols; it does not produce in ten years as many deaths as it formerly caused in one, and, I believe, never rages in them as a contagious epidemic. Yet it is evident from the facts already stated, that both fevers and bowel complaints are, even at the present period, much more frequent causes of death in prisons than amongst the general population. How is this to be explained?

The Reports of the Prison Inspectors contain ample evidence of the prevalence of bowel complaints in many of the County Gaols and Houses of Correction. They also prove that it is generally ascribed to some peculiarity of the prison dietaries. Great difference of opinion, however, seems to prevail as to the particular article or quality of diet which is in fault. In one prison the greater prevalence of the disease in the summer season is ascribed to the use of *old* potatoes; in another, to the use of *new* potatoes. In a third, the gruel is thought to be the *cause*; while in a fourth, a bason of gruel at night is given as a *preventive*. The most generally-received opinion, however, I believe to be, that the *liquid* nature of the diet is the cause of the complaint, and several facts strongly favour this view of the matter.

In the first place it is certain that many prisons liable to the prevalence of diarrhœa have sites which are not *manifestly* insalubrious; and it has further been observed, that while the disease has prevailed in a prison, the population of the immediate neighbourhood has not suffered from it in any remarkable degree. Hence the inference would appear obvious, that the cause of the disease was one to which the prisoners alone were exposed; and in the absence of any other evident source of the evil, it would be natural to ascribe it to the diet; for if food of a liquid form were capable of producing diarrhœa, such an effect might certainly be expected to result from the dietaries which have, until recently, been in use in many prisons. It has happened moreover, on several occasions, that an unusual prevalence, constituting almost an epidemic of diarrhœa, has followed a reduction of the nutritious quality of the diet, and has either ceased, or has become mitigated, on the diet being again improved. These facts certainly favour the opinion that the diarrhœa of prisons is produced by the liquid quality of the food.

But there are other facts which prevent my concurring in this view. If it were the correct one, diarrhœa should have been prevalent in all prisons in which the food was principally liquid. This has by no means been the case. There are many prisons in which this complaint has been of rare occurrence, although the dietary has consisted chiefly of liquids. I need instance merely the Salford and Preston Houses of Correction, where bowel complaints have been very infrequent, although for se-

veral years the prisoners were allowed less solid meat and bread than in other prisons where diarrhœa was prevalent, and much more liquid food, namely, six pints of soup and twenty-eight pints of gruel weekly.

Besides, we can scarcely conceive that a merely liquid and unstimulating diet, though it might produce simple looseness of the bowels, could give rise to acute inflammation of the large intestines, or dysentery. Yet, wherever diarrhœa is very prevalent, this more severe form of the disease also shows itself occasionally. In many of those instances, too, in which more solid and nutritious food has produced so beneficial an effect, the disease has not wholly disappeared. It has been much mitigated in severity, and has become less extensively prevalent, but it has still continued to be more frequent amongst the prisoners than amongst the population of the country around.* Lastly, the officers, and the families of those resident in the

* A remarkable exemplification of this statement is afforded by the history of the epidemic which prevailed in the Millbank Penitentiary during the year 1823. The epidemic broke out shortly after the diet of the prisoners had been rendered very meagre. The diet was improved, and immediately the severity of the disease became mitigated. The conclusion to be drawn from these facts seemed obvious; and the physicians appointed by Government to investigate the cause of the disease, reported that it had been produced by the reduction of the diet, conjoined with the cold of the preceding winter. But soon the renewed violence of the epidemic, while the diet was abundant, and while the weather, with the advance of spring, was becoming milder, taught them that their judgment had been an erroneous one.

prison, have generally not escaped, but on the contrary have occasionally suffered very severely, especially at those times when the disease has been most prevalent amongst the prisoners.

For these reasons, it seems to me impossible to regard the diet as the sole cause of the prevalence of diarrhœa in prisons. There are other facts, which, besides being inexplicable according to that view, seem to indicate, in a positive manner, that the disease really depends on some form of malaria floating in the atmosphere.

One fact of this kind is, that the diarrhœa of prisons varies in its degree of prevalence according to the season and the state of the atmosphere, just as the more severe dysentery does in countries where that disease is confessedly dependent on the influence of miasm. In dry, frosty weather, in winter, it is of rare occurrence; in the spring it is more frequent if the weather be moist as well as mild; and its prevalence is usually greatest in the latter part of the summer, and in the autumn, especially if great heat has succeeded to rain, or if rain has succeeded to hot and dry weather. In those years, too, when bowel complaints are unusually prevalent in the town or country in which a prison is situated, they manifest themselves with disproportionate severity amongst the prisoners.

This has recently been exemplified in the Millbank Penitentiary. During the year 1842, bowel complaints were unusually prevalent in the metropolis, and the mortality produced by them

was twice as great as in either of the two previous years. In the same year, dysentery prevailed to a great extent in the Penitentiary, and in a few months was the cause of as many deaths as all forms of bowel complaints had given rise to during the seventeen previous years.

These are not the only grounds for believing that bowel complaints, when they prevail in prisons through a series of years, are due to the influence of malaria. I have visited many prisons in different parts of England, and have met with none in which the prevalence of bowel complaints was remarkable, and the site, at the same time, free from obvious sources of malaria. Either the particular locality was low and damp, or there was moist, imperfectly-drained land, with more or less of stagnant water in the immediate vicinity, or the whole tract of country was relatively low, imperfectly drained, and subject to inundations in rainy seasons, the soil clayey and damp, and the atmosphere subject to fogs.* Where the prevalence of diarrhœa in prisons was not so great, there these characters of the locality existed in a less marked degree. The land, perhaps, was not at all too damp for the purposes of agriculture, but from some cause or other was generally in a moist state: either it re-

* Such, for example, is the situation of the Wakefield House of Correction. In the second report of the Inspector for the northern and eastern district, it is described as standing "in a low humid situation, on the margin of a water-course." It is surrounded by extensive meadows, which have a stiff clay soil, and are flooded in the winter.

tained for a long period the rain which fell upon it, or it was kept moist by land springs. Where the disease was of rare occurrence, the situation of the prison was eminently salubrious. The same rule will be found to hold good with regard to lunatic asylums,* workhouses and barracks. In the last-mentioned establishments, the production of diarrhœa by the poorness of diet is out of the question; the rations of soldiers in quarters in England are abundant, and everywhere the same. Yet there are barracks distinguished by the prevalence of diarrhœa. This was the case in a remarkable degree two or three years ago at the Tower. At the Portman-street Barracks, on the contrary, where the soldiers' lodgings were quite as ill-constructed, diarrhœa had been a very rare ailment. No obvious cause for this

* I would instance the Lunatic Asylums at Hanwell and Wakefield, as illustrations of this remark. The former institution has a site most favourable to health. The latter, on the contrary, lies in a hollow, surrounded by fields and gardens, which have a sub-soil of clay, and are generally damp; while close to it, and partly encompassing it, is a plantation which grows out of a damp moat-like excavation. With respect to these two Asylums, Sir William Ellis, who in both had held the office of medical superintendent, makes the following remarks in his work on Insanity and Lunatic Asylums:—"Independent of diseases peculiarly connected with the nervous system, the insane seem particularly subject to others, such as chronic inflammation of the bowels, diarrhœa and dysentery. These diseases appear to depend a good deal on locality; in the Asylum at Wakefield, a large proportion of the deaths was at one time owing to them; whilst in the one at Hanwell, they are comparatively of rare occurrence; this possibly may be accounted for by the former being on a cold clay soil, and the latter on a fine bed of gravel."

difference in the health of the two establishments existed, except the dissimilarity of their localities, and especially the presence of a moat, half choked up with mud and weeds, which then surrounded the Tower Barracks.*

We have seen that the theory which ascribes the production of diarrhœa to the poorness and liquid nature of the prisoners' food will not account for all the facts of the case. Now in this respect, the other explanation, namely, that the disease is caused by miasms floating in the atmosphere, has a great advantage. For, adopting this view, we shall still be able to understand how the nature of the diet should have great influence on the prevalence of the disease. The increased prevalence of the bowel complaints consequent on the reduction of the diet, is, indeed, no more than we should expect; for if the atmosphere were slightly impregnated with malaria, it might be incapable of affecting prisoners while their systems were supported by even a moderate allowance of food, and yet might produce serious effects on them when they had been weakened by low diet. For the same reason, persons living in the immediate neighbourhood, and even the officers residing within the prison walls, may escape the attacks of the disease altogether, or suffer much less frequently than the prisoners, because, being better fed, and perhaps fortified by the use of fermented liquors of some

* The moat surrounding the Tower has, within the last year or two, been drained and partly filled up, but sufficient time has not yet elapsed since these measures were completed, to allow of our drawing conclusions as to their effects on the health.

kind or other, they are better able to resist the influence of the slightly poisoned air which they breathe; while the prisoners, being deficient in nervous vigour and activity of the nutritive processes, in consequence of scanty diet, confinement and more or less of mental depression, are extraordinarily susceptible of any noxious influence. A parallel case, is that of armies encamped in malarious countries. It is a well known fact, that, under these circumstances, the officers either escape altogether, or suffer in a far less degree than the men, from the endemic disease.*

The foregoing remarks have reference to the various forms of bowel complaints prevalent in prisons. The origin of the fever is a still more difficult subject of investigation. But my belief is, that many of the cases of fever, like the bowel complaints, are really of endemic or malarious origin. I of course admit that fever, like diarrhœa, may occur in prisons from other causes than malaria. Persons confined in prisons which are situated in large and closely-populated towns or cities, like Liverpool or London, will be liable to infection, when an epidemic typhus prevails amongst the surrounding population. But in such instances as those of the Wakefield House of Correction, in this country, and the Sing Sing Prison of the American State of New

* See Pringle's Observations on the Diseases of the Army, 5th edition, 1765, p. 67 and 183; and Tulloch's Reports on the Sickness, Mortality and Invaliding among the Troops in Western Africa, p. 25; in the Mauritius, p. 22 c.; and in the West Indies, p. 97.

York, where fever has prevailed at intervals for a series of years, concurrently or alternately with dysentery and diarrhœa, and where no immediate contiguity of a dense population exists to explain its origin, it seems to me impossible to doubt that the fever, as well as the bowel complaints, has been caused by malaria generated in the localities. Moreover, the fever which has occasionally shown itself in the Millbank Penitentiary differs in some of its most striking features from the continued fever, or typhus, of crowded cities, as well as from the "gaol fever" of former periods, and approaches more nearly to certain forms of the "remittent fever" of malarious countries.*

If the views here proposed be correct, great practical benefits would arise from the adoption of the

* The drinking of bad water is one of the reputed causes of dysentery and fever. Yet there seems to be very little evidence to show that an endemic prevalence, of either of these diseases, has ever arisen from such a cause. That the bowel complaints so common in English prisons, workhouses and lunatic asylums do not depend on the quality of the water, I am perfectly satisfied. In several of these institutions, in which bowel complaints are most rife, the water used for drinking is obtained from deep wells, and cannot, therefore, contain any putrescent organic matter. And in the Millbank Prison it has recently been proved in a conclusive manner that the Thames water, which the prisoners drink, does not cause the diarrhœa from which they frequently suffer. In consequence of it having been suggested to one of the Inspectors of that prison, that the Thames water, cleansed by subsidence only, and not by filtration, was likely to exert a deleterious influence on the health of the prisoners, a supply of the purest water from a neighbouring Artesian well was obtained every second day for two months; and one third of the prisoners (about 350)

following preventive means: 1. Previous to the erection of any new prison, an elevated dry site should be most carefully selected. 2. Wherever a prison already built is liable to the prevalence of fever or bowel complaints, all obvious sources of malaria should at once be got rid of, and the drainage of the surrounding land should be made as perfect as possible: if there should be any very low and damp spots, these should be filled up; and the land, if in cultivation, should be laid out in such crops as require least moisture. 3. As there are, perhaps, localities, where the development of malaria, owing to a peculiarity of the soil and substrata, can be prevented by no practicable system of drainage, it will in some instances be necessary to give the prisoners a more liberal diet than would be required in other gaols, in order that they may resist the influence of the miasm still floating in the atmosphere.

Returning from this digression, I will now briefly recapitulate the principal results of the inquiry, respecting the influence of unhealthy sites, on the mortality of English prisons.

It was assumed, as the basis of this investigation, that any increase of the mortality of a prison produced by insalubrity of site, would be shown in the number of deaths from fevers and bowel complaints.

were supplied with this water only, whilst the other two thirds (about 700) were allowed to drink the Thames water as before. The result was, that no difference in the health of the two bodies of prisoners was experienced. Proportionally as many cases of diarrhœa, and also of other diseases, occurred amongst the one as amongst the other.

We sought therefore to determine from statistical data, first, whether the mortality from these diseases in particular prisons had much exceeded the average mortality produced by them in prisons generally; and secondly, whether fevers and bowel complaints had been much more fatal to criminals confined in prisons, than to all persons at the same period of life, living in the metropolis. The facts which we examined with the view to determine the latter point, placed it beyond a doubt, that fevers and bowel complaints had been between four and five times as fatal in the county prisons of England, taken collectively, as in the metropolis generally. And the result of our comparison of the number of deaths caused by these diseases in different prisons, showed that they had not been equally prevalent, or at least, not equally fatal in all prisons, since there were some of these establishments in which the mortality from fevers and bowel complaints had been much below the average, and others in which it had considerably exceeded the average; the excess being greatest in the case of the Wakefield House of Correction. We found, too, that in the Millbank Penitentiary, the mortality from these diseases, though greater than the average mortality caused by them in the county prisons taken collectively, yet had been less than in the Wakefield House of Correction, and one or two other prisons.

If now we place side by side the rates of mortality due to fevers and bowel complaints, and the rates of mortality from all causes in the Millbank Peniten-

tiary, the Wakefield House of Correction and the thirty-two other county prisons, we shall see that the total mortality, both in the Millbank Penitentiary and in the Wakefield House of Correction, has been much above the average total mortality of the other prisons, but that in the first-named prison the diseases which can be ascribed to the influence of malaria have produced a very small part of the excess in the total mortality, while, in the case of the Wakefield House of Correction, that class of diseases has been the principal source of the increased ratio of deaths.

	Total mortality per 1,000 prisoners.	Mortality from fevers and bowel complaints per 1,000 prisoners.
32 County prisons (those included in Tables XX. and XXI. with the exception of the Wakefield House of Correction)	18·597	3·412
Millbank Penitentiary (123 of the pardons being added to the total mortality, and one-fourth of the pardons on account of fevers and bowel complaints, to the mortality from those diseases)	30·965	5·944
Wakefield House of Correction	26·732	8·910
Excess of mortality in the Millbank Penitentiary above the average of 32 county prisons	12·368	2·532
Excess of mortality in the Wakefield House of Correction, above the average of 32 county prisons	8·135	4·498

The average annual mortality per thousand prisoners from all causes, in the Penitentiary, (making the addition previously explained for the pardons on medical grounds,) has exceeded, by more than twelve (12·368) deaths, the total average annual mortality of thirty-two county prisons, calculated

only from the deaths actually occurring in those prisons. And the deaths caused by fevers and bowel complaints will account for only 2·532 out of that annual excess of twelve deaths. On the other hand, the total annual mortality in the Wakefield House of Correction (without any allowance for pardons on medical grounds) has exceeded the average annual mortality of the thirty-two other county prisons, by rather more than eight (8·135) deaths per thousand prisoners, and of this excess 4·498 deaths per thousand prisoners, or more than half, have been due to fevers and bowel complaints.

The remaining excess of mortality in the Wakefield prison, amounting to 3·637 deaths per thousand prisoners annually, seems to have been owing, not to any greater prevalence of the diseases, produced or aggravated by imprisonment, but solely to the circumstance, that the practice of recommending prisoners in ill-health for pardon on medical grounds has been adopted to a less extent in the Wakefield House of Correction than in other county prisons.*

* During the last two or three years, at all events, the pardons have been proportionally less numerous in the Wakefield prison than in other prisons, as will be seen in the subjoined Table :—

Prisons.	Liverpool.	Lancaster Castle	Chester Castle.	Derby.	Preston.	Wakefield.
Years.	1841 and 1842.	1841, 1842 and 1843.	1842 and 1843.	1842 and 1843.	1841, 1842 and 1843.	1842, and 1843.
Number of deaths	16	9	6	4	1	25
Number of pardons	13	8	4	3	2	5

It appears, then, that the rate of mortality of the Wakefield House of Correction has been raised above the average rate of other English prisons, chiefly if not exclusively by a noxious influence of the locality, while the influence of the site has had but a slight share in augmenting the number of deaths in the Millbank Penitentiary; the high rate of mortality in this institution having been due to causes more immediately connected with the state of imprisonment.

Rates of Mortality of the Criminal Class, in Prison and out of Prison.—We have now considered the influence of the several circumstances which although, as it were, extraneous to the state of imprisonment, and not conditions necessarily or constantly attending it, yet influence greatly the number of deaths occurring in prisons.

Some of the facts which we have passed in review have an important bearing on the question which we previously left unanswered—the question, namely, whether the comparatively high rate of mortality common to most prisons can be ascribed to the unhealthiness of the degraded class whence criminals are in greater part derived—whether, in fact, the mortality of prisoners is only the mortality proper to persons whose constitutions have been injured by the combined influences of destitution and debauchery.

We have seen that the real mortality, at least of the English prisons, is by no means wholly exhibited in the number of deaths actually occurring in these prisons, but that the number of prisoners

pardoned on account of the bad state of their health must also be taken into the account; and we have found that, according to a fair estimate of the proportion of cases that would have terminated fatally amongst the whole number pardoned on medical grounds, the rate of mortality of the Millbank Penitentiary would have been 30·976, and that of the county prisons 22·788, per thousand prisoners. These are higher rates of mortality than we can suppose the criminal class, out of prison, to experience; and consequently these high rates of mortality alone form a ground for believing that the mortality of criminals is increased by imprisonment.

But we have further seen that the rates of mortality are, *cæteris paribus*, highest in those prisons where the terms of imprisonment are longest, and lowest where the terms of imprisonment are shortest; and likewise that the mortality to which the prisoners are liable, is far less in the earlier periods than in the more advanced periods of their imprisonment in the same prison. Now if the high rate of mortality of prisons were wholly or even principally due to the unhealthiness of the criminal class, we should not find that the increased duration of imprisonment was thus attended by a higher ratio of deaths.

This fact, indeed, can only be explained on the supposition that the state of imprisonment, or conditions attending it, have exerted an injurious influence on the prisoners' health. The *low* rate of mortality during

the first six months of the prisoners' confinement is doubtless in part owing to the circumstance that criminals, when they enter prison, are for the most part free from acute disease, and even from very advanced chronic disease. It can, however, be scarcely necessary to observe that this is not the sole nor even the chief cause of the difference between the mortality of earlier and later periods of imprisonment. For, amongst prisoners in the more advanced periods of their term of imprisonment, the proportion of deaths is twice or even three times as great as amongst any class of persons in the general population of the same age as prisoners. Liverpool presents a higher rate of mortality than any other town in England, and its population, including as it does a large number of Irish, is probably even more unhealthy than the whole body of criminal prisoners in England; for the prisoners come from all ranks of society, and from all districts of the country—from the rich as well as the poorest classes—from the agricultural as well as the manufacturing districts; yet the rate of mortality of the population of Liverpool, between the ages of 15 and 70, is much lower than that of prisoners even in the county prisons of England, and consequently very much lower than the rate of mortality which the prisoners in the Millbank Penitentiary suffered, especially at the more advanced periods of their terms of confinement. This is shown in the following Table :—

Annual mortality of the population of London between the ages of 15 and 70 during 1841	15·391 per 1,000 living.
Annual mortality of the population of Liverpool between the ages of 15 and 70 during 1841	18·191 per 1,000 living.
Estimated average annual mortality of prisoners in the county prisons of England, including a certain proportion ($\frac{346}{1000}$) of the pardons on medical grounds	22·788 per 1,000 prisoners.
Estimated annual mortality of prisoners in the Millbank Penitentiary, including a certain proportion ($\frac{346}{1000}$) of the pardons on medical grounds	30·976 per 1,000 prisoners.
Estimated rate of mortality of the prisoners in the Millbank Penitentiary, during the third year of their imprisonment	52·267 per 1,000 prisoners.

The mortality of the population between the ages above mentioned, was, in the year 1841, 15·391 per thousand in London, and 18·191 per thousand in Liverpool. The mortality of the county prisons of England has been 22·788 per thousand; that of the Millbank Penitentiary, at all periods of imprisonment, nearly 31 per thousand, whilst the estimated rate of mortality of convicts passing through their third year of confinement in that institution was more than 52 per thousand.*

The statistics of the American prisons may, at first sight, seem to prove that the state of imprisonment has not everywhere been attended by an in-

* Instances may be met with in which the mortality of the inhabitants of a particular town, or part of a town, has exceeded the mortality of the population of other towns, or other parts of the same town, to a greater extent than the mortality of Liverpool is in the text stated to have exceeded that of London. For exam-

creased mortality. The white convicts in these prisons certainly appear to have suffered much lower rates of mortality than prisoners in England and on the continent of Europe. For the proportion of

ple, the mortality in two different groups of streets in Brussels, during the three years 1840 to 1842, was, according to M. Ducpétiaux (Des Décès dans la Ville de Bruxelles, p. 14 to 18), as follows :—

In 5 streets in which more than half of the families are poor, - - -	The mortality was 33 per 1,000 persons living.
In 250 streets in which there were no poor families, - - -	The mortality was 20 per 1,000 persons living.

It is to be recollected, however, that in this instance, and others of the same kind, we have presented to us the mortality of persons of all ages. And not only is the mortality in the early years of life always much higher than in after years, but, moreover, the increase of mortality which so often attends poverty falls chiefly on the very young, and not nearly so much on persons of the middle period of life. Thus, in the instance above given, 551 out of 1,000 deaths occurred during the first 5 years of life in the poorer streets of Brussels, while only 320 out of 1,000 occurred in these early years in the richer streets. So that if these early years of life were excluded from consideration, the difference between the mortality of the poorer and that of the richer class would be much less than the difference between 20 and 33 per 1,000 living. In fact, the mortality at all ages in Liverpool was greater than in the poorest streets of Brussels, although, between the ages of 15 and 70, it was only 18 per 1,000 annually.

	London.	Liverpool.
Mortality at all ages	24·269	33·882
Mortality from 15 to 70	15·390	18·191

deaths amongst the white convicts in the Eastern Penitentiary, during a series of years, has been only 20 per thousand annually, while in the County Prisons of England, the proportion of deaths has been nearly 23 per thousand, in the Millbank Penitentiary 31 per thousand, in the Geneva and Lausanne Penitentiaries respectively 26 and 38 per thousand, and in the French prisons (males) 55 per thousand. This comparatively low rate of mortality of the white convicts in the Eastern Penitentiary, appears the more remarkable from the circumstance that the average term of imprisonment is longer in that institution than in any of the prisons with which it is here compared, except those of France. It is true the rate of mortality (20 per 1,000 annually) assigned to the Eastern Penitentiary, being deduced solely from the number of deaths which have actually occurred in the institution, is somewhat below the true rate of mortality. For the pardoning of prisoners, whose health had become impaired, has doubtless reduced the number of deaths in the American prisons, as it has in those of England. The number of pardons on medical grounds in the Eastern Penitentiary, however, appears to have been so inconsiderable, that, had no pardons been granted, the whole mortality would probably not have been greater than that of the English County Prisons, namely, 23 per thousand annually, and this would have been a very low rate of mortality if we consider the average length of the sentences in the Eastern Penitentiary. In other American prisons, the par-

dons on medical grounds have probably been more numerous than in that institution; but, on the other hand, the deaths in those prisons have been fewer; so that we cannot certainly assign to the Auburn, Charlestown, and Wethersfield prisons a higher rate of mortality than the one just mentioned, namely, 23 deaths annually per thousand prisoners. The causes of the comparatively low ratio of deaths amongst the white convicts in the American prisons will be fully considered when we come to speak of the prevalence of scrofulous disease in different prisons, and of the causes to which that prevalence is due. I will here, therefore, merely mention one cause which has undoubtedly been highly influential in preserving the health of prisoners in American penitentiaries; namely, their very abundant and nutritious food. We shall find that the liberal diet of American prisoners contrasts strongly with the meagre allowance to which, until recently, criminals in most of the English County and Borough Prisons were restricted, and still more with the even scantier fare of imprisoned criminals in Scotland.

Admitting, however, that the proportional number of deaths is smaller amongst white convicts in American prisons, than amongst the criminals in the prisons of this and other countries of Europe, we are still able to show that imprisonment, even in America, is attended with an increase of mortality. We have previously seen that the annual mortality of the white population of the City and County of New York, between the ages of 10 and 60, is scarcely

13 per thousand of the living, that the annual mortality of the same population above the age of 10 years is about 15 per thousand living, and that the mortality of the white male population alone in the City of New York, between the ages of 10 and 70 years, is likewise about 15 per thousand annually. Now, the question is, whether either of these proportional numbers can be taken to represent the rate of mortality to which the criminals of the white race in the American States are subject when not in prison, or whether we must assign to them as high a rate of mortality as we have done to the criminal class in England. The following considerations lead me to believe that the rate of 15 deaths annually per thousand living may, for the purpose of comparison with the mortality of white prisoners in the American Penitentiaries, be fairly taken to represent the mortality of this class of persons when at liberty.

In the first place, it must be borne in mind that the lowest class of society in the American States, especially in those which, like Pennsylvania, border on the slave states, is formed chiefly of coloured people, and that the whites are for the most part not exposed to the same degree of destitution, and such strong temptations to brutal vice and debauchery, as the lowest class of society in England, whence our criminals are chiefly, though not wholly, derived. On this account it is probable that but little difference exists as regards health between the white criminals in America before their imprisonment, and the general white population of

such a City as New York. Secondly, we should remember that in taking as our standard of comparison the mortality of the whole white population of New York above the age of 10 years, or even the males between the ages of 10 and 70, we are including a large number of persons at an advanced period of life, who, by reason of their age, are subject to a very great mortality, while in prisons there are few persons older than 60 or 65 years.

Lest, however, any one should still think that the mortality of the white population of New York, of the ages mentioned, namely, 15 deaths annually per thousand living, is somewhat lower than the mortality to which criminals even at liberty would be subject, we will assume the ratio of deaths in the American prisons to have been only 20 per thousand annually, which is the rate of mortality of white prisoners in the Eastern Penitentiary, calculated from the deaths alone, without including any cases which were prevented from terminating fatally in the institution by the criminals being pardoned. According to this estimate, the annual mortality suffered by white persons in America will have been raised from 15 per thousand to 20 per thousand by the influence of imprisonment, and even this must be regarded as a considerable increase in the ratio of deaths amongst persons who are chiefly in the meridian of life.

With regard to the coloured persons, the increased mortality which *they* suffer from imprisonment is almost unparalleled; the mortality of these people, out of prison, being 27·225 annually

per thousand living of both sexes, (29·115 per thousand of the males,) and in prison as much as 70·10 per thousand. A large number of the negroes, it is true, have been in a diseased state when admitted into the penitentiaries; but the extraordinary mortality they have suffered while in prison cannot be wholly accounted for by this circumstance. It seems, indeed, to be generally admitted in America, that imprisonment exerts a most injurious influence on the health of coloured persons.

Both in England and in America, therefore, imprisonment seems to have been productive of a great increase of mortality; and looking at the high rates of mortality of the prisons of France and Switzerland, we cannot doubt that the same has been the case in those countries. This fact, then, being established, we have next to inquire what the disease or diseases are to which the increase of mortality is chiefly due; for till this point is determined, we cannot usefully investigate the effects of the several circumstances capable of influencing the health, to which criminals are exposed in the state of imprisonment.

III.—Diseases by which the Mortality of Prisons is chiefly produced.

Fatal Diseases of the Prisoners in Millbank Penitentiary.—In the investigation of the rate of mortality in prisons, we first examined the statistics of the Millbank Penitentiary, and afterwards compared the results they afforded with data derived from

other sources. It will be convenient to follow the same plan in the present inquiry respecting the diseases which have been most fatal to prisoners.

We have already determined what amount of mortality has been caused in the Millbank Penitentiary, and the other English prisons, by three classes of diseases, viz. fevers, bowel complaints and hepatic diseases. We have found that the mortality from hepatic diseases has been less in these prisons than in the metropolis, but that fevers and bowel complaints have been four or five times more fatal to prisoners than to all persons at the same period of life in London. We have also found that the proportional number of deaths caused by fevers and bowel complaints *in the Millbank Penitentiary*, though somewhat greater than *in most County Prisons*, has not been so much in excess, as to account for the higher rate of mortality by which the Penitentiary is distinguished from most of the latter prisons. We will now direct our attention more especially to the rates of mortality due to fevers, bowel complaints and hepatic diseases, *in the Millbank Penitentiary* and *in the metropolis respectively*, and will compare these rates of mortality with each other, in order to determine in what degree the greater mortality of the *prisoners in the Penitentiary*, as *compared with persons at liberty*, has been caused by these diseases, and what excess in the ratio of deaths remains to be explained by other causes, when the mortality from fevers and bowel complaints has been deducted.

	Annual mortality per 1,000 from fevers.	Annual mortality per 1,000 from bowel complaints.	Annual mortality per 1,000 from hepatic diseases.	Annual mortality per 1,000 from these 3 classes of diseases.
Millbank Penitentiary (including one fourth of the cases pardoned on medical grounds)	3·702	2·242	·156	6·100
Metropolis 1842*	·552	·538	·327	1·418
Excess of mortality in the Millbank Penitentiary	3·150	1·704		4·682

From this table it appears that the total excess of mortality from the three classes of diseases, fevers, bowel complaints and hepatic diseases, in the Millbank Penitentiary, as compared with the metropolis, has been 4·682 deaths per thousand annually. The mortality from all causes in the Penitentiary, however, we have estimated at 30·976 deaths per thousand annually, while the mortality from all causes, amongst persons of the ages of 15 to 70 in the metropolis, has been only 14·707. In the total mortality, then, there has been an excess of 16·269 deaths per thousand annually, on the side of the Penitentiary, and fevers and bowel complaints have produced little more than one-fourth of this excess. There remain 11·587 deaths annually per thousand prisoners to be accounted for by other causes.

The mortality caused by the different classes of diseases (exclusive of fevers, bowel complaints and hepatic affections) in the Millbank Penitentiary and in the metropolis respectively, is shown in the next table; where the diseases most fatal to prisoners

* See the note (†) at p. 171.

may be distinguished at a single glance. The class of "epidemic diseases, exclusive of fever, diarrhœa, dysentery and cholera;" the "diseases of the head and nervous system," and the "abdominal diseases (exclusive of enteritis and ulceration of the bowels)," have, each of them, produced a rather greater mortality in the Penitentiary than in the metropolis generally; the excess of mortality produced by the "diseases of undefined nature" is somewhat more considerable; but the whole excess of mortality from these four classes of diseases amounts to no more than 2·322 deaths per thousand annually; and this is entirely compensated for by the deficiency in the mortality caused by "heart diseases," "diseases of the respiratory organs (exclusive of consumption and hæmoptysis)," "diseases of the urinary and generative apparatus," the "diseases not classified," and "violent deaths," which have produced fewer deaths in the Penitentiary than in the metropolis generally. The great excess of mortality, it is manifest, has been caused by "consumption and hæmoptysis," and "other tubercular diseases." The annual ratio of deaths from consumption and hæmoptysis I have computed to have been 13·244 per thousand in the Penitentiary, while it has been only 4·374 in the metropolis. The annual mortality from other tubercular diseases, according to my estimate, has been 2·869 per thousand in the Penitentiary, and only ·033 per thousand in the metropolis. So that tubercular diseases have produced in the Penitentiary an excess of mortality equal to 11·706 deaths per thousand annually,—which is nearly exactly the number of

deaths by which the mortality of the prisoners in the Penitentiary has exceeded the total mortality of persons of corresponding ages in the metropolis, after the deaths caused by fevers, bowel complaints and hepatic diseases have been deducted.

	Annual mortality per 1,000 persons living, from										
	Epidemic diseases, not including fevers and bowel complaints	Consumption and hæmoptysis.	Other tubercular or scrofulous diseases.	Diseases of the head and nervous system.	Diseases of heart and circulating system.	Diseases of the respiratory organs, exclusive of consumption.	Abdominal diseases not including fluxes.	Diseases of the urinary and generative apparatus.	Other diseases, not classified.	Diseases of undefined nature.	Suicide and violent deaths.
Deaths in the Penitentiary.	·834	7·613	·835	·730	·104	·625	·312	·000	·417	·730	·313*
Cases pardoned, which would have terminated fatally.	·000	5·631	2·034	·808	·500	·365	·261	·287	·704	1·773	
Total estimated mortality of the Penitentiary.	·834	13·244	2·869	1·538	·604	·990	·573	·287	1·121	2·503	·313
Mortality between the ages of 15 and 70 in London.	·213	4·374†	·033	1·411	·627	2·106	·305	·534	1·820	1·195	·581‡
Excess of mortality in the Penitentiary.	·621	8·870	2·836	·127	—	—	·268	—	—	1·306	
Excess of mortality in the metropolis.	—	—	—	—	·023	1·116	—	·247	·699	—	·268

This result is so remarkable, and its accuracy depends so much on the correctness of the estimate I have formed of the mortality produced by different

* Suicides alone.

† Including half the deaths from hæmorrhage, which we may suppose to have been caused by hæmoptysis.

‡ Various kinds of violent deaths.

diseases in the Penitentiary, that some explanation of the data and method of computation on which this estimate is founded seems to be called for. Now, with regard to the deaths which actually occurred in the prison, no doubt can be entertained. The annual mortality per thousand caused by each disease, or each class of diseases, has been calculated from the number of deaths during the 18 years, 1825 to 1842, and from the aggregate of the average numbers of prisoners in confinement during those several years. Thus, the number of deaths from consumption and hæmoptysis, namely 73, has been multiplied by 1,000, and then divided by 9,588, the aggregate of the numbers in prison in the 18 several years (see Table XVIII. in the Appendix). The quotient, 7·613, was, of course, the annual ratio of deaths per thousand prisoners which consumption and hæmoptysis had produced during the whole period. The calculation of the number of deaths caused within the prison itself by each class of diseases is, therefore, a mere matter of arithmetic.

It is not so with the estimate of the mortality which the pardons on account of each disease should be taken to represent. Here we have to exercise our judgment upon the fatal tendency of the different diseases, and upon their probable rate of progress towards a fatal termination in persons confined in prison. For, as the prisoners pardoned on medical grounds appear to have been released, on the average, about one year before the expiration of their sentence, the question is, how many of the prisoners pardoned on account of each disease, or class of diseases, would have died

before the expiration of a twelvemonth if they had remained in prison.

From my knowledge of the usual rate at which cases of consumption proceed to a fatal termination in the state of imprisonment, and of the period of the disease at which prisoners suffering from it have in most cases been pardoned, I have thought it by no means an exaggerated estimate, to set down three-fifths of the pardons, granted on account of consumption and hæmoptysis, as deaths,—that is to say, I have supposed that 54 out of the 90 prisoners pardoned on account of those diseases would have died in the prison, had their confinement been prolonged until the expiration of their sentences. In the case of diseases of the heart likewise, I have reckoned that three-fifths of the pardons (4·8 out of 8) may be regarded as deaths. But with respect to all other diseases, I have assumed that in only one out of every four of the cases where they formed the grounds of pardon (that is to say, in 64·25 out of the 257 cases), would the prisoner have died in prison, supposing that no remission of his sentence had been granted. If the list of diseases in Table XIX. be examined, it will be admitted, I think, that this is a just estimate. For in a large number of cases the diseases were such as either do not usually prove fatal, or do so only after a very long course; in confirmation of which remark I may instance the pardons on account of chronic rheumatism, ulcers of the legs, epilepsy, and uterine disorders. The grounds of pardon in a very large number of cases are recorded as general ill health, debility, and vis-

ceral disease. It is difficult to form a decided opinion as to what proportion of those cases would have proved fatal. It is most probable, however, that the majority of the more serious of them really belonged to the category of scrofulous or tubercular disease of the lungs, or other internal parts, and, consequently, when we estimate the proportion of fatal cases to have been only one in four amongst the pardons on account of these diseases of undefined nature, we are underrating the mortality from consumption rather than the mortality from other causes. With regard to the 78 cases in which scrofula formed the ground of pardon, we cannot doubt that in many of these also the lungs were seriously affected with scrofulous or tubercular disease; but as, in many others, the chief development of the disease must have been external, I have estimated the proportion of fatal cases to have been only one-fourth of the whole number.*

Without any exaggeration then of the facts, it seems to me to be clearly proved by the medical records of the Penitentiary, that the mortality caused by tubercular disease has been between three and four times as great during the eighteen years, 1825 to 1842, among the convicts confined in this prison, as it was in the year 1842 amongst persons of the same period of life in London generally; and that three-fourths of the excess of deaths from all causes in the Penitentiary above the rate of mortality of all

* It is in accordance with the estimate here given of the probable fatal tendency of the different diseases which have formed the grounds of pardon, that the number of deaths represented by the 355 pardons on medical grounds is stated at the commencement of this paper to be 123.

persons in the metropolis of the same period of life, has been due to the prevalence of this disease; fevers and bowel complaints having produced only one-fourth of that excess of deaths.

The facts and arguments have already been detailed on which I found my belief that the high rate of mortality suffered by prisoners in the Millbank Penitentiary cannot, except in small part, be ascribed to the unhealthiness of the criminal class. This opinion will be seen to be confirmed by the following statistical facts.

In the course of the year 1840, 1,052 prisoners were received into the Penitentiary, and in twelve out of this number, signs of phthisis were detected, either at the time of their reception, or so soon afterwards as to leave no doubt as to the disease having existed, although in a latent state, when they were received into the institution. There is every reason for supposing that the remaining 1,040 prisoners were free from the disease.

Now of these 1,040 convicts, only 523 were Penitentiary prisoners, who were confined for periods varying from one to four years, but on the average for about two-and-a-half years, while 517 were convicts under sentence of transportation, who passed a much shorter time in the Penitentiary—on the average, not more than two months. Amongst the latter class, no further development of phthisis was observed, but of the 523 Penitentiary prisoners, no less than forty-seven came under treatment for pulmonary phthisis, and seventeen died of that disease before the end of 1843.

Comparing the large number of these prisoners in whom tubercular disease of the lungs first showed itself while they were in the Penitentiary, with the small number who were affected with it at the time of their reception, we cannot, I think, hesitate to admit that imprisonment exerted here a very powerful influence in causing the development of the disease.

I have said, that twelve out of 1,052 prisoners received in the year 1840 (1·141 per cent.) were phthisical at the time of their reception; but it is to be remarked, that in more than half of these cases, the disease was then latent. The proportion of cases of phthisis existing in an *active state*, amongst criminals when they enter prison, is, I believe, more accurately shown by the following data, extracted from the Medical Register of the male convicts received into Millbank Prison during the year 1844. The total number of male convicts registered was 3,249. The number of phthisical persons amongst them was eleven, and their proportional number, consequently, only 3·38 per thousand.

From these facts it must, I think, be obvious, that the proportion of persons affected with phthisis amongst the criminals received, has been far too small to account for any considerable part of the great mortality which that disease every year produced amongst the prisoners confined in the Penitentiary.

Fatal diseases in the English County Prisons.—On account of the comparatively small number of prisoners in the individual county prisons, no conclusion could be fairly drawn from the number of deaths from different diseases in each of these

prisons taken separately. I shall therefore examine here merely the data afforded by the mortality tables of thirty-three county prisons taken collectively. From these tables it appears (see the Abstract table below*) that consumption and other tubercular diseases have not produced the same excessive mortality in the county prisons as in the Millbank Penitentiary, and that a proportionally greater amount of mortality has been caused, not only by fevers and bowel complaints, but also by several other classes of disease,—especially by the sporadic diseases of the respiratory organs, not registered as of consumptive nature.

	Annual mortality per 1,000 persons living, from													
	Fevers.	Other epidemic diseases.	Consumption and hæmoptysis.	Other tubercular or scrofulous diseases.	Diseases of the brain and nervous system.	Diseases of the heart and circulating system.	Diseases of the respiratory organs, exclusive of consumption.	Hepatic diseases.	Bowel complaints.	Diseases of the abdomen, exclusive of bowel complaints, enteritis and ulceration.	Diseases of the urinary and generative apparatus.	Other diseases not classified.	Diseases of undefined nature.	Suicide and other violent deaths.
33 English county prisons.	2·386	·407	4·540	·232	2·270	·291	3·376	·261	1·833	36	·291	1·251	1·688	·724
Persons aged from 15 to 70 in the metropolis.	·552	·213	4·374	·033	1·411	·627	2·106	·327	·538	·305	·534	1·820	1·195	·581
Excess of mortality in the county prisons.	1·834	·194	·166	·199	·859	—	1·270	—	1·295	·131	—	—	·493	·143
Excess of mortality in the metropolis.	—	—	—	—	—	·336	—	·066	—	—	·243	·569		

It must be remembered, however, that this table

* The Tables of which this is an abstract, are those numbered XX. and XXI. in the Appendix.

exhibits the rates of mortality calculated solely from the deaths which actually occurred in the prisons, and that the cases of prisoners pardoned in a diseased state are taken no account of. In a former part of this paper, I have shown that the proportion borne by the pardons on medical grounds to the deaths, in the county prisons, has been such, that supposing the number of fatal cases amongst these pardons to have been the same in the county prisons as they appear to have been in the Penitentiary, the mortality of these prisons should be estimated at twenty-three deaths, instead of nineteen deaths, per thousand prisoners annually. By a similar calculation we may form an approximative estimate of the mortality which would have arisen from consumption and other tubercular diseases in these county prisons, had no prisoners been pardoned on account of impaired health. The result of such a computation is given in the next Table; and it will be seen that even with this correction, the excess of mortality caused by consumption and scrofula has been less than that produced by fevers and bowel complaints taken together.

	Mortality per 1,000 from consumption and hæmoptysis.	Mortality per 1,000 from other tubercular or scrofulous diseases.	Mortality per 1,000 from consumption and all tubercular diseases.
County Prisons	6·257	·858	7·115
Metropolis	4·374	·033	4·407
Excess of mortality in the County Prisons.	1·883	·825	2·708

Although, therefore, consumption and other forms

of scrofula have been more fatal to the prisoners confined in the county gaols and houses of correction of England, than to the inhabitants of the metropolis, they have produced much less mortality in those prisons than in the Millbank Penitentiary. This result, however, by no means militates against the belief that the tubercular cachexia, most frequently fatal, in the form of pulmonary phthisis, is the disease which imprisonment has an especial tendency to produce. For, as I have before pointed out, the terms of imprisonment which persons undergo in the county prisons, are, in a vast majority of cases, so short, that if the germs of the tubercular phthisis were formed during the state of imprisonment, there would not be time for the disease to become fully developed, much less for it to reach its fatal termination before the prisoner's discharge. Owing to this circumstance, we should expect that the mortality from consumption would be much less considerable in the county prisons than in the Millbank Penitentiary. Indeed, all circumstances being considered, it appears to me that the amount of mortality from consumption in the county prisons has been quite sufficient to prove that imprisonment in those establishments has the same tendency to produce tubercular disease as confinement in the Penitentiary. The average term of imprisonment in the county prisons being under two months, while some prisoners are detained as long as eighteen months or two years, a very large proportion of the terms must have been under one month.

Now it is a very rare occurrence for a prisoner to die of consumption within a month, or even two months, after being committed to prison. The deaths from this disease must, therefore, have occurred principally amongst the prisoners confined for long terms, and their number being comparatively small, the mortality from consumption amongst them must have been excessive.

I shall have a future opportunity of demonstrating the correctness of this inference, when the influence of length of imprisonment on the development of tubercular disease shall come more especially under consideration. In the meantime, however, I may mention a fact which proves clearly enough that prisoners confined for short periods have suffered even less mortality from tubercular disease in the Penitentiary than in the county prisons. Of 3,592 military offenders who, in the course of the five years, 1838 to 1842 inclusive, were confined in the Penitentiary for various periods, and whose average term of imprisonment was two months and five days, only one died of consumption. Two others, affected with this disease, were pardoned before the expiration of their sentences. But even if these two cases are reckoned as deaths, which they should not be, the annual rate of mortality from consumption will have been only 4·601 per thousand upon the average number of soldiers in confinement.

Fatal diseases in the English Hulks.—I am not able to give any account of the diseases which have

been fatal to the convicts in the English Hulks. This, however, is the less to be regretted, since no inference as to the effects of imprisonment in those establishments on the health could be deduced from the mortality caused there by different diseases; circumstances already explained, having led to the accumulation in the Hulks, of prisoners who had become diseased in other prisons.

Fatal diseases in the Prisons and Hulks of France.—Satisfactory statistical information respecting the diseases fatal in the prisons and hulks of France is also wanting. Some reports, however, which have been published by the Physicians of the Maisons de Correction at Nismes and Rennes, show that tubercular consumption has been the principal fatal disease in those prisons, as it has been in the Millbank Penitentiary.

M. Toulmouche, Physician to the prison at Rennes, places phthisis first in the order of frequency amongst the causes of death, and states, moreover, that in the course of three years, (1831 to 1833,) when the average number of prisoners was 540, no less than 238 cases of pulmonary phthisis, and 31 cases of glandular enlargement, came under medical treatment.* And in the prison at Nismes, out of twelve deaths, which occurred in the wards of M. Boileau Castelneau during the year 1838, six were due to phthisis, and two to other tubercular diseases.†

* Annal. d'Hygiène, tom. xiv., p. 1, et seq.

† Annal. d'Hygiène, tom. xxii., p. 207. The causes of death

Fatal diseases in the Prisons of Switzerland.—The statistics of the Geneva and Lausanne Penitentiaries are much more complete. And although, owing to the number of prisoners in these institutions being small, and the deaths also few, any inferences from the data they afford would not alone have much weight, yet they serve to confirm the accuracy of the conclusions deduced from other facts.

In the Geneva Penitentiary, the annual mortality from consumption has been 9·892 per thousand prisoners, and the mortality from other scrofulous or tubercular diseases 2·198 per thousand. The annual mortality from all tubercular diseases has therefore been 12·090 per thousand, while the annual ratio of deaths from all causes has been only 26·380 per thousand. Now, if it be borne in mind that we have no account of the number of prisoners pardoned in consequence of their being in an impaired state of health, and also that the average terms of imprisonment have been considerably shorter in the Geneva Penitentiary than at Millbank, it will, I think, be admitted that the mortality from tubercular disease has been, proportionally, quite as great in the former as in the latter institution.

In the Lausanne Penitentiary, the annual ratio of

in the twelve cases are recorded as follows :—Two cases of scrofula with phthisis, one of scrofulous ophthalmia with phthisis, two of white swelling with phthisis, one of phthisis, one of scrofula with chronic pericarditis, one of external scrofula with fever, one of abscess of the iliac fossa, one of disease of the cervical vertebræ, one of white swelling, and one after the operation of lithotomy.

deaths recorded as due to consumption has been only 6·802 per thousand prisoners. One-fourth of the total number of deaths has been ascribed to "marasmus," which, in the words of M. Gosse,* was "probably complicated with disease of the lungs;" and if we suppose only half of these cases (which in the Table are referred to the diseases of undefined nature) to have been really of the nature of pulmonary consumption, the mortality from this disease will have been 11·337 per thousand annually.

	Annual mortality per 1,000 living, from											
	Fevers.	Consumption.	Other tubercular diseases.	Diseases of the brain and nervous system.	Diseases of the heart.	Diseases of the respiratory organs, exclusive of consumption.	Bowel complaints.	Other diseases of the abdomen.	Diseases of the urinary and generative apparatus.	Diseases not classified.	Diseases of undefined nature.	All causes.
Geneva Penitentiary†	1·099	9·892	2·198	5·496	·000	4·396	1·099	1·099	·000	1·099	·000	26·38
Lausanne Penitentiary‡	·000	6·802	·000	5·669	2·267	2·267	2·267	—	1·134	5·669	10·204	36·28

The mortality from diseases of the brain and nervous system appears to have been very considerable, both in the Geneva and in the Lausanne Penitentiaries. But we must not hence infer that imprisonment in those institutions has a peculiar tendency to produce diseases of that class; for where the number of prisoners is small, the occurrence of two or three cases more than ordinary, of any spora-

* Examen Médicale et Philosophique du Système Pénitentiare. Geneva, 1838, p. 198.

† See Table XXXIII. in the Appendix.

‡ See Table XXXIV. in the Appendix.

dic disease will cause the mortality from that disease to appear unusually great; and the high ratio of deaths from diseases of the brain in the Geneva and Lausanne Penitentiaries, is, without doubt, a circumstance of this accidental nature. The same explanation applies to the high rate of mortality from the group of diseases not classified in the Lausanne Penitentiary.

Fatal Diseases in the American Prisons.—I proceed now to show the amount of mortality produced by consumption and other forms of tubercular disease amongst prisoners in America. The terms of confinement in the American prisons have been for the most part even longer than in the Millbank Penitentiary; and the official reports of the more important of these establishments, many of which I have been enabled to consult, contain accurate information as to the diseases fatal to the prisoners. We have here then presented to us the means of determining, in a tolerably satisfactory manner, whether imprisonment for several months or years has in other prisons, besides the Millbank Penitentiary, produced the same prevalence of tubercular disease—whether, in other prisons, as well as in the Millbank Penitentiary, the greater mortality of prisoners, as compared with persons at liberty, has been chiefly, indeed almost exclusively, due to tubercular phthisis. The data are deficient only as regards the pardons granted to prisoners on account of impaired health, respecting which we have no satisfactory information.

We will confine our attention at first to the *white convicts* in the American State prisons; for a part of the official documents before referred to, afford data from which we can determine the mortality caused by different diseases amongst the white and the coloured convicts separately. The Statistical Tables of the Eastern Penitentiary of Pennsylvania give, not only the number of deaths amongst the white and coloured convicts respectively in that institution, but also specify the nature of the disease which was fatal to each individual prisoner during several years. In the Auburn and Charlestown prisons, too, the proportion of coloured prisoners to the whole number in confinement has been so small that we cannot be far wrong in taking the mortality from different diseases amongst the whole body of prisoners to represent the rate of mortality suffered by white convicts from those diseases. In the following Table, therefore, the ratio of deaths from different diseases, and classes of diseases, amongst the white convicts in the Eastern Penitentiary,* and amongst all convicts in the Auburn and Charlestown State Prisons,† during a series of years, is compared with the mortality which the same diseases produced amongst the white male population of the city and county of New York, during the year 1842.‡

* See Table XXVII. in the Appendix.

† See Tables XXX. and XXXI. in the Appendix.

‡ See Table XXXV. in the Appendix.

	Annual mortality per 1,000 living, from														
	Fevers.	Epidemic diseases exclusive of fevers and bowel complaints.	Consumption and hæmoptysis.	Other tubercular diseases.	Diseases of the brain and nervous system.	Diseases of the heart and circulating system.	Diseases of the respiratory organs, exclusive of consumption.	Hepatic diseases.	Bowel complts.	Other abdominal diseases.	Diseases of the urinary and generative apparatus.	Other diseases not classified.	Diseases of undefined nature.	Suicides and violent deaths.	Total mortality.
White convicts in the Eastern Penitentiary.	·677	2·031	12·864	·000	1·354	·677	·677	·000	·677	·000	·677	·677	·000	·000	20·311
All convicts in the Auburn State Prison.	1·099	·244	9·526	·366	·977	·244	2·687	·366	·610	·732	·122	·977	·488	·977	19·418
All convicts in the Charlestown State Prison.	1·961	·000	10·787	·000	·980	·326	1·634	·000	·653	·000	·000	1·307	1·306	·653	19·614
White male population of New York.	1·380	·778	4·706	·167	2·167	·472	1·389	·315	·935	·241	·129	·778	·231	1·288	15·054

The results of this comparison are, 1st, That no class of diseases but that of consumption and hæmoptysis has produced, uniformly in the three prisons, a greater mortality than in the city and county of New York ; 2nd, That fevers, other epidemic diseases, diseases of the brain and nervous system, diseases of the heart and vascular system, hepatic affections, bowel complaints, other abdominal dis-

eases, diseases of the urinary and generative apparatus, and violent deaths, have produced a rate of mortality below the standard of comparison, if not in the three prisons, at least in two out of the three; 3rd, That there has been a slight excess of mortality from the group of unclassed diseases in the Auburn and Charlestown prisons, and an excess equal to 1·298 death per thousand persons living, from the sporadic diseases of the respiratory organs in the Auburn prison; but, 4th, That the annual mortality per thousand persons living, from consumption and hæmoptysis, amongst the white convicts, has exceeded the rate of mortality from the same causes amongst the white male inhabitants of New York, by as much as 8·158 deaths in the Eastern Penitentiary, by 4·820 deaths in the Auburn prison, and by 6·081 deaths in the Charlestown State prison. From other forms of tubercular disease no deaths appear to have occurred either in the Charlestown prison, or amongst the white convicts in the Eastern Penitentiary, and only a trifling mortality in the Auburn prison.

Now the mortality from consumption and other forms of tubercular disease in the Millbank Penitentiary, has exceeded the mortality from the same causes in London, by as much as 11·706 deaths per thousand prisoners annually. It would appear, therefore, that the excess of mortality from this disease had been less considerable in the American prisons than in the English Penitentiary. It must be remembered, however, that in calculating the

mortality of the Penitentiary at Millbank, a certain proportion of the pardons on medical grounds was added to the deaths from each disease, while in the case of the American prisons the estimate of the mortality is formed solely from the deaths which actually occurred in the prison. This is one reason why the excess of deaths from consumption and other tubercular diseases does not appear so great in the latter prisons as in the former. Another reason is, that the white criminals in America, as I have already pointed out, are originally, in all probability, a healthier class of persons than the criminals in England. But we must still, I believe, admit a third reason, namely, that imprisonment in most of the American Penitentiaries has really exerted a less injurious influence on the health than confinement in the Penitentiary of Millbank. Into the various causes of this difference I shall inquire on a future occasion. For my present purpose it is only necessary to show that the whole excess of mortality amongst the convicts, as compared with persons at liberty, has been due to tubercular diseases, as well in the American prisons as in the Millbank Penitentiary.

This fact is exhibited most clearly in the annexed tabular view of the rates of mortality from consumption and other tubercular diseases, and from all other sporadic diseases, in the different prisons of England and America, and in the cities of London and New York. Epidemic diseases are not included, because they vary so much in their degree of prevalence in different pri-

sons, from merely accidental circumstances, and quite independently of the influence of imprisonment.

Cities and Prisons.	Annual mortality per 1,000 living, from		
	Consumption, hæmoptysis and other tubercular diseases.	All other sporadic diseases.	All causes, fevers, other epidemic diseases and bowel complaints excepted.
Millbank Penitentiary	16·113	8·083	24·196
English County Prisons	4·772	10·395	15·167
Inhabitants of London between the ages of 15 and 70	4·407	8·997	13·404
Eastern Penitentiary of Pennsylvania, (White Convicts)	12·864	4·062	16·926
Auburn State Prison	9·892	7·565	17·455
Charlestown State Prison	10·787	6·213	17·000
White male population of New York, between the ages of 10 and 70	4·873	7·088	11·961

Directing our attention first to the county prisons of this country, we see that in these establishments, where the terms of imprisonment are generally short, the excess of mortality above the rate to which persons in the metropolis are subject, has not been greater in the case of the tubercular, than in that of other sporadic diseases.

Now, we ought to obtain the same result on comparing the American prisons with the city and county of New York, if prolonged imprisonment in those institutions had no greater tendency to generate tubercular phthisis than to cause other sporadic diseases. We find, however, that in re-

gard to the relative mortality from tubercular and other sporadic diseases, the American prisons resemble the Millbank Penitentiary much more than the English County Gaols and Houses of Correction. For while the mortality from other sporadic diseases has been less in the Charlestown Prison and the Eastern Penitentiary, and only a fraction greater in the Auburn Prison,* than in the city and county of New York, the mortality from consumption, and other tubercular diseases, has been more than twice as great amongst the convicts in the Auburn and Charlestown Prisons, and three times as great in the Eastern Penitentiary, as amongst the white male population of New York. In the Millbank Penitentiary the proportional mortality from tubercular diseases has been between three and four times as great as the proportional mortality from the same diseases in the metropolis; and the ratio of deaths from other sporadic diseases has been $\frac{1}{8}$th higher in the metropolis than in the Penitentiary.

There is yet another way in which we may demonstrate that consumption and other diseases of the same nature have been the predominant causes

* The disease which has produced the greatest excess of mortality next to consumption, in the Auburn State Prison, is inflammation of the lungs. And the number of the fatal cases of this disease has been so much greater in the Auburn, than in the other prisons, that I cannot help suspecting that in many of the cases the diseases were of tubercular nature, and not simple pneumonia.

of death amongst the white convicts in the American prisons, as well as amongst the criminals in the Millbank Penitentiary. It consists in showing the proportion borne by the deaths from those diseases, to the whole number of deaths from all sporadic diseases in the prisons of England and America, and in the cities of London and New York. This is done in the table at p. 217, where it will be seen that the proportion of the total mortality from sporadic diseases which has been caused by tubercular complaints, has been in

London	$\frac{1}{3}$rd	or	$\frac{5}{15}$ths.
The English County Prisons	$\frac{1}{3}$rd	or	$\frac{5}{15}$ths.
Millbank Penitentiary .	$\frac{2}{3}$rds	or	$\frac{10}{15}$ths.
City and County of New York, rather more than	$\frac{2}{5}$ths	or	$\frac{6}{15}$ths.
Auburn State Prison, nearly	$\frac{3}{5}$ths	or	$\frac{9}{15}$ths.
Charlestown State Prison, rather more than	$\frac{3}{5}$ths	or	$\frac{9}{15}$ths.
Eastern Penitentiary, nearly	$\frac{4}{5}$ths	or	$\frac{12}{15}$ths.

In whichever way, then, we regard the facts, it is equally apparent that the tubercular cachexia has been the predominant cause of death amongst prisoners (of the white race) confined for long terms, in America as well as in England, and that when epidemic and endemic diseases are excluded from consideration, the whole excess of mortality amongst prisoners, as compared with the free white inhabitants of an American city, may be ascribed to tubercular disease.

Now, when we inquire respecting the causes of death amongst criminals belonging to the *dark races*,

in the American prisons, we meet with proofs that consumption and other forms of tubercular scrofula have been even more destructive to this class of prisoners than to the white convicts. The only accurate information which we possess respecting the diseases fatal to blacks in the state of imprisonment is derived from the official reports of the Eastern Penitentiary of Pennsylvania.* But the results deducible from the facts there given are entirely confirmed by the Mortality Tables of the Maryland Penitentiary.† For in the latter institution more than half the prisoners are Negroes or Indians; and it will be seen from the following Table that the mortality from consumption has been extraordinarily great.

	Annual mortality per 1,000 living, from				
	Consumption, hæmoptysis and other tubercular diseases.	Other sporadic diseases.	All sporadic diseases.	Fevers, bowel complaints and other epidemic diseases.	All diseases.
Coloured prisoners in the Eastern Penitentiary of Pennsylvania.	40·748	25·321	66·069	3·303	69·372
All prisoners in the Maryland Penitentiary.	28·490	8·547	37·037	1·709	38·747 ‡
Coloured male population of New York above the age of 10 years.	11·011	13·626	24·637	4·478	29·115 §

* See Table XXVIII. Appendix. † See Table XXXII. Appendix.

‡ Not including Asiatic cholera.

§ Different classifications of the ages of the coloured people being

The rate of mortality from consumption and other tubercular diseases has been 11 per thousand annually amongst the coloured male population of the City of New York; while in the Eastern Penitentiary it appears to have been as high as 40 per thousand; and even this is, I believe, below the truth. For it will be seen, on referring to Table XXVIII. in the Appendix, that out of the total number of 63 deaths amongst the coloured convicts in the Eastern Penitentiary, 13 are ascribed to chronic inflammation and disorganization of the lungs, and we can scarcely doubt that many of these were really cases of tubercular disease. This opinion derives confirmation from the circumstance that no other sporadic diseases but those of the respiratory organs appear to have produced an excessive amount of mortality amongst the coloured people in the Eastern Peni-

adopted in the Census of the United States, and in the Tables of Mortality, we cannot ascertain the rate of mortality to which that part of the population is subject between the ages of 10 and 70. To compare the deaths with the population, it has been necessary to include all ages above 10 years. The rate of mortality has been calculated from the Census of 1840, and the deaths in 1842, and therefore is probably not quite correct. The error, however, cannot be great, for it will be seen by Table XXXVI. in the Appendix, that the rates of mortality from all causes and from tubercular diseases are found to be very nearly the same, whether the deaths in 1838 or the mean of the deaths in 1838 to 1842 be compared with the population of 1840. It would seem, in fact, that the black population of the State of New York does not increase.

tentiary, and also from the fact that in the Maryland Penitentiary, where more than half the convicts are blacks, the excess of mortality has been wholly due to consumption, only one death having been caused by any other disease of the respiratory organs (see Table XXXII. in the Appendix).

There is one other American State Prison, namely, that of Sing Sing, near New York, the statistics of which afford tolerably complete information as to the diseases causing death amongst the convicts. I have not hitherto made any reference to the data derived from this source, in the present inquiry respecting the mortality caused by tubercular disease amongst prisoners; because the deaths appertaining to the coloured prisoners are there not distinguished from those of the white prisoners; and because the proportion of Negroes and Indians amongst the whole body of prisoners (about $\frac{1}{5}$th) is too small to render the liability of criminals of the dark races to particular diseases very perceptible in the tables of mortality, and yet so large as to prevent our inferring from those tables, the frequency of a particular cause of death amongst the white convicts. The statistics of the Sing Sing Prison are valuable, however, from the light they throw on another question of some interest,—the question, namely, whether or not the presence in the atmosphere, of malaria, arising from the soil, has a tendency to produce tubercular disease, and to increase the mortality from the more fatal forms of that malady. We have previously

seen that the mortality from fevers, bowel complaints, (especially dysentery,) and hepatic diseases, has been so considerable in the Sing Sing Prison, as to place it beyond a doubt that the site is a malarious one. Now, when we inquire as to the mortality caused by tubercular diseases in this prison, we find that it has been very little greater than in the Auburn and Charlestown prisons, where the diseases which can be ascribed to the influence of malaria have produced few deaths, and that it has been actually less than amongst the white convicts in the Eastern Penitentiary, who likewise have suffered but little from diseases referrible to malaria.

	Annual mortality per 1,000 living, from				
	Consumption and other tubercular diseases.	Fevers and other epidemic diseases, bowel complaints and hepatic diseases.	All other sporadic diseases.	All diseases, exclusive of fevers, other epidemic diseases, bowel complaints, hepatic diseases and Asiatic cholera.	All diseases except Asiatic cholera.
Male population of New York, aged 10 years and upwards.	5·304	3·562	7·763	13·067	16·629
Auburn State Prison.	9·892	2·329	7·197	17·089	16·418
Charlestown State Prison.	10·787	2·614	6·213	17·000	19·614
Eastern Penitentiary, white convicts.	12·864	3·385	4·062	16·926	20·311
Sing Sing State Prison.	11·265	9·277	9·835	21·100	30·377

The mortality from "other sporadic diseases" has been considerably greater in the Sing Sing Prison than in the other state prisons; but on examining the list of fatal diseases in the former prison, we find that this excess of mortality has been owing, not to any disease which we can suppose to have been of tubercular nature, but to dropsy, which has caused a large number of deaths, and in all probability has been in great part produced by malarious influence. There seems every reason, therefore, to believe that the annual ratio of deaths caused by tubercular disease has been really no greater in the State Prison of Sing Sing than in other American Penitentiaries. This result is not an unimportant one. For it is strong evidence of the incapability of malaria to produce tubercular disease,* and, as we shall presently see, affords a valuable ground of argument in support of the position that the locality of the Millbank Penitentiary has had only an inconsiderable share in producing the high rate of mortality amongst the prisoners in that institution.

The fatal diseases in the Millbank Penitentiary the same, and due to the same causes, as in other prisons.—In a previous part of this paper, it was

* At the same time it may be remarked, that the amount of tubercular disease among the prisoners in the Sing Sing State Prison has been far too considerable to afford any support to the opinion recently put forth by several French writers, that phthisis is of rare occurrence in malarious countries,—that malaria in fact impedes the development of the disease,—an opinion which seems to be based on very imperfect statistical data.

shown that the diseases which from their nature might be ascribed to the operation of malaria, had produced but a small part of the mortality of the Penitentiary at Millbank. Owing, however, to the exaggerated notion of the insalubrity of the locality which has prevailed, many persons would probably still imagine that the noxious local influence, although it did not occasion great mortality from those diseases which are generally recognised as the effects of malaria, yet might increase the mortality from other diseases. The facts detailed in the foregoing pages seem to me to demonstrate quite satisfactorily the erroneousness of such an opinion, which, moreover, has not one positive argument in its favour. It has been shown that the increased mortality in the Millbank Penitentiary has been due, almost wholly, to the diseases which are characterized by the deposition of tubercular matter in different organs of the body, but principally in the lungs; and it has further been shown that in all prisons where convicts have been confined for long terms, the same state of things has prevailed; that in Switzerland, and in America, as well as in the Millbank Penitentiary, tubercular diseases have produced a rate of mortality twice or three times as high as the same diseases cause amongst the free population of cities; while the mortality from no other diseases or class of diseases has uniformly in all prisons been raised above the ordinary ratio amongst the free population.

From these facts the obvious conclusion seems to be, that an increased amount of tubercular disease has been the general result of imprisonment, and that the causes of the mortality in the Millbank Penitentiary are not to be sought for in circumstances peculiar to that institution, but rather in conditions generally attendant on the punishment of imprisonment as hitherto carried out.

The rate of mortality from tubercular diseases appears to have been lower, it is true, in most prisons than in the Millbank Penitentiary, but the reason of this has been, in some cases, the shorter duration of the terms of imprisonment, and in others probably the apparent reduction of the mortality caused by the prisoners being pardoned on account of ill health; an addition having been made to the mortality from tubercular diseases, so as to compensate for this source of error, in the case of the Penitentiary, but not in the case of other prisons. Still it must be admitted, that, as far as the published facts show, the white prisoners in America appear to have suffered less mortality from tubercular disease, and consequently less mortality altogether, than the prisoners in the Millbank Penitentiary. Now the case of the Sing Sing Prison is valuable, inasmuch as it shows that the greater mortality from consumption, and other tubercular diseases in the Millbank Penitentiary, cannot be ascribed to the influence of malaria exhaled from the surrounding grounds, or from the banks of the river. For at Sing Sing the influence of malaria

has been manifested in other ways much more unequivocally than at Millbank, yet the mortality from tubercular diseases appears to have been no greater in the Prison of Sing Sing than in other American prisons, which are governed by the same rules, and have salubrious sites.

The site of the Millbank Penitentiary, it cannot be denied, is so far calculated to promote the prevalence of tubercular disease, as it is in close proximity to a most densely populated part of a large city; for the air of crowded cities is, doubtless, more favourable to the development of that disease than is the air of an open country. But the influence of this circumstance must have been comparatively trifling. The causes which operated most powerfully in the production of tubercular disease in the Millbank Penitentiary, were, I believe, as in other prisons, wholly independent of the locality.

The fact that tubercular phthisis is the most frequent cause of death in prisons has been noticed by several continental writers; but it has not hitherto, as far as I know, been demonstrated by a sufficient amount of evidence of a precise character, and has not attracted much attention. That phthisis should be the disease most fatal to prisoners could not indeed appear at all remarkable, since we know that amongst the general population at the middle period of life there is no other cause of death which produces nearly so great a mortality. In order that the fact should have much importance

or interest, it was necessary to show from trustworthy statistical data, that the mortality from consumption has borne a larger proportion to the whole number of deaths, and also to the number of persons living, in prisons than in society at large; and to do this has been one of my principal objects in the present paper.

Prevalence of scrofula in prisons.—It is however the prevalence in prisons, not of pulmonary phthisis merely, but of all forms of the scrofulous cachexia, characterized by the deposition of tubercular matter, which I have wished to illustrate. The most frequent and chief seat of the disease, when it proves fatal, being the lungs, and the published reports on prisons seldom giving exact information with regard to the prevalence of any diseases but those causing death, the statistical facts which I have been able to collect, tend in most instances to prove only that great mortality has arisen from *phthisis pulmonalis*. Yet I am not the less convinced that other forms of tubercular disease, and especially disease of the external absorbent glands (or scrofula), have been proportionally quite as prevalent.

In the case of the Millbank Penitentiary, this admits of being satisfactorily demonstrated. In the first place it will be seen, on examining the list of pardons on medical grounds, that in 78 out of 337 cases, scrofula formed the ground of pardon. It would of course be only in aggravated cases that the existence of scrofula would be considered a ground for releasing a convicted criminal from imprison-

ment; we may therefore infer that the prevalence of the disease must indeed have been very great, if in the course of eighteen years, 78 cases occurred in which release from imprisonment seemed called for.

But I am able to adduce still stronger evidence of the prevalence of tubercular disease in the Penitentiary. During the year 1840, 1,052 prisoners were received into the Penitentiary. At the time of their reception, fourteen of these prisoners, or 1·330 per cent., had scrofulous enlargement, with or without suppuration, of the cervical glands, combined in four cases with the signs of pulmonary phthisis; and 1,038 were to all appearance free from external scrofulous disease. Of these 1,038 prisoners, 527 were Penitentiary prisoners, and 511 were females under sentence of transportation, who passed a much shorter time in the Penitentiary. Of the latter class of prisoners, two became affected with scrofula while in the Penitentiary; but of the former class, no less than thirty-seven had, before the end of 1843, come under treatment for external glandular scrofula, which was combined in fourteen cases with tubercular disease of the lungs. This fact, that 37 out of 527 persons became affected with scrofula in the course of so short a period, will surely be admitted as sufficient evidence of imprisonment in the Penitentiary having had a tendency to produce other forms of tubercular disease besides pulmonary consumption.

From the data just detailed, it appears, that the proportion of scrofulous individuals amongst

the prisoners received into the Millbank Penitentiary, in the course of the year 1840, was 1·330 per cent. In order to determine whether this can be taken as the average proportion of cases of scrofula amongst criminals in England, before they have undergone long terms of imprisonment, I have examined the Medical Register of Convicts received into the Millbank Prison since it has been converted into a depôt for transports. The state of health of 3,249 male convicts received during the year 1843 is there accurately recorded, and forty-four, or 1·354 per cent., are set down as being affected with external scrofula.* This result corresponds very closely with that afforded by the statistics of the year 1840: we must therefore, I think, regard it as tolerably certain that about $1\frac{1}{3}$rd per cent. of the criminals sent to the Millbank Penitentiary, or Prison, are affected with external scrofula.

Of those who are scrofulous when received at this institution, there are, however, many in whom the disease has first shown itself while they were in the county prisons, consequently the proportion of scrofulous individuals amongst criminals who have as

* The proportion of criminals bearing cicatrices of scrofulous sores on their necks, amongst those received at the Millbank Penitentiary, is also shown by the Medical Register. It appears that out of 3,249 male convicts received into the prison during 1843, 27, or 8·31 per thousand, had such scrofulous cicatrices. I am not acquainted with any facts which, serving as standards of comparison, would enable us to determine whether this proportion is a very large one.

yet suffered no imprisonment must be even less than 1⅓rd per cent.

With respect to the influence of imprisonment in the county prisons, I feel satisfied that if the terms of imprisonment had been of equal duration, a far greater amount of scrofulous disease would have been produced in them than in the Millbank Penitentiary; and I base this opinion on the following grounds,—first, the large proportional number of cases of scrofula amongst those prisoners who had been confined six or eight months in county prisons, before they were sent to the Penitentiary; secondly, the fact, that at least half the prisoners who were scrofulous when received, declared the disease to have first shown itself during their confinement in the county prisons; and thirdly, my knowledge of the degree in which prisoners in these establishments have been exposed to the causes of scrofula.

I regret that I am at present unable to give any statistical information as to the prevalence of scrofula amongst the prisoners in Scotland, and can only state generally, that the proportion of scrofulous individuals has been far greater amongst the convicts sent to Millbank from the prisons of Scotland, than amongst those who come from the poorest districts of England.

In the Penitentiary of Lausanne, the chief diseases of the prisoners are stated by the physician, Dr. Pellis, to be a kind of chlorosis, affecting both sexes, and the *tubercular cachexia*; and the latter disease is said to affect not only the lungs, but also the intestines, the *glands of the neck*, the mem-

branes of the brain and the limbs, with a relative frequency corresponding with the order in which these parts are here enumerated.*

In proof of the prevalence of scrofulous disease in American prisons, I need only direct attention to the list of the causes of death amongst the black convicts in the Eastern Penitentiary (see Table XXVIII.), where it is shown that in fifteen out of sixty-three fatal cases, the prisoners were the subjects of scrofulous disease in other parts of the body, besides the lungs, and to the following remarks of the physicians of two of the Penitentiaries of the United States.

Dr. Bache, Physician of the Eastern Penitentiary of Pennsylvania, states that "the predominant diseases in that prison were *scrofulous diseases*, dyspepsias and affections of the chest, including *consumption*." Dr. Coleman, the Physician of the New Jersey Penitentiary, says, "the tendency to glandular obstruction is seen in almost every prisoner who has been confined in the cells for more than a year, when he is in the least degree indisposed. * * * Some *post mortem* examinations have been made, and in all of them the lymphatic glands were enlarged to an enormous degree, indurated and obstructed." And in a subsequent report:—"The greater number of cases on the (sick) list, are those forms of debility that depend on glandular obstructions, and seem peculiar to prisons."

* See Varrentrap's paper, "Die Schweizer Straf-anstalten," in the Jahrbücher der Gefängnisskunde, bd. i. p. 8.

The remarkable prevalence of scrofulous disease amongst the black convicts in the American prisons, is certainly owing to their being extremely liable to the disease, and very frequently affected with it at the time of their entering the Penitentiaries. But with respect to the white convicts in the United States, it appears that the prevalence of scrofula amongst them, before they have undergone imprisonment, is rather less than amongst the criminals admitted into the Millbank Penitentiary. Such, at least, is the only inference which can be drawn from the following facts:—During the two years 1838 and 1839, 160 white convicts and 93 blacks were received into the Eastern Penitentiary. Of the blacks eight, but of the whites only two, were scrofulous. In the year 1842, 102 whites and 40 blacks were received, and amongst the whole number there were three affected with scrofula. Now, if we suppose two of these three to have been blacks, and one a white convict, the proportion of scrofulous patients amongst the two classes received in the three years will have been only 1·145 per cent. for the whites, and 7·519 for the blacks.

The foregoing facts, relative to the prevalence of external glandular scrofula amongst criminals, are adduced as supplementary evidence in support of the main position advanced in the latter part of this paper. It was there shown that the increased mortality attending the state of imprisonment is almost entirely due to the greater number of deaths caused by internal tubercular disease, and especially by

pulmonary phthisis. We now see that tubercular disease of the external glands, also, is very frequent amongst criminals in confinement, while it is by no means so common amongst the same class of persons at liberty; we seem, therefore, to be justified in the conclusion that it is not merely pulmonary phthisis, but tubercular disease in all its forms, which is so general a result of the long-continued influence of imprisonment on the bodily health.

The length to which this communication has already extended, obliges me to refrain from further recapitulation of the facts which I have sought to establish. For the same reason I must reserve the investigation of the *causes* on which the great mortality from tubercular diseases in prisons has depended, for the subject of a second paper, and will here merely state that the most influential of those causes appear to have been,—1st, deficient ventilation; 2nd, cold; 3rd, want of active bodily exercise, and sedentary occupations; 4th, a listless, if not dejected state of mind; and 5th, poorness of the diet,—for the diet of prisons, though often perhaps more abundant than the agricultural labourer usually enjoys, yet has generally been less stimulating, and also less nutritious, than seems to be requisite for the health, under conditions so unnatural and depressing as are those almost necessarily attendant on the state of

imprisonment. There are one or two other points which deserve investigation, namely, the influence of sex and age in modifying the liability of prisoners to suffer from tubercular cachexia, and the periods of imprisonment at which the first symptoms of the disease become apparent, and at which its development is completed: these, also, are topics which must be discussed on a future occasion.

One remark, however, must be added here, in order to prevent misconception of the purport of the present paper. The facts which I have detailed are not intended to authorize, and indeed do not justify, any sweeping condemnation of imprisonment as a system of secondary punishment. For, although during the fifteen or twenty years preceding 1843, prisoners confined for periods of two, three, or four years have generally suffered a much greater mortality than persons of the same period of life out of prison, it does not follow that such will always be the effect of confinement in prisons. Many of the causes which have been most active in the production of the fatal disease of prisoners, such as cold, scanty food and confined air, have no necessary connection with the state of imprisonment. And already, in fact, as I have before mentioned, a beginning has been made in the work of freeing this mode of punishment from most of the injurious conditions which have hitherto generally attended it. In several prisons newly built, the inmates are protected against all, or almost all, those influences which have the greatest tendency to injure the bodily health, and in

many others very important improvements have been effected. These changes cannot fail to produce a good result; and, if the statistics of penal establishments should be again collected and analysed at the end of the ensuing ten years, it will, we cannot doubt, be found that the mortality of the prisoners has been considerably lowered, and the ravages amongst them, of tubercular scrofula especially, in a great measure mitigated.

Postscript.—While the preceding pages have been passing through the press, the author has met with a paper on the Prisons of Norway, by Professor Holst. (Om Sygepleien i Straffeanstalterne i Norge; aftryk fra Norsk Magazin for Lægevidenskaben. Christiana, 1841.) It there appears that the annual mortality has been only 17·646 per thousand amongst the convicts in the fortresses (Slaverier), and 16·823 per thousand in the Houses of Correction (Tugthuse) of Norway. Moreover, it would seem that these low rates of mortality are chiefly due to the small number of deaths from consumption; the ratio of deaths from this disease having been only 3·135 per thousand convicts in the Fortresses, and 2·692 in the Houses of Correction; whilst, among all persons of the ages of 15 to 70 in London, the annual mortality, as we have seen, is 4·374 per thousand. If these data could be relied on, they would lead us to infer, either that confinement in the ill-regulated prisons of Norway actually diminished the mortality of a disease, whose ravages have been greatly increased by imprisonment in other countries, (a supposition which can scarcely for a moment be entertained,) or that the climate of Norway itself is unfavourable to the development of tubercular disease. There is, however, much reason to suspect that the returns obtained by Dr. Holst from the different prisons were far from accurate.

TABLE I.—Showing the number of prisoners received into the Millbank Penitentiary in each year from 1824 to 1842 inclusive, the daily average of prisoners in confinement during each year, and the number of deaths, pardons on medical grounds, and removals to the Hulks for the benefit of the health.

Years.	Received during the year.				Average daily number.			Deaths.			Pardons on medical grounds.			Ratio per 1,000 of deaths to the average number of prisoners.			Ratio per 1,000 of pardons on medical grounds.			Ratio per 1,000 of deaths and pardons.			Sent to the Hulks for benefit of health.
	Penitentiary prisoners.		Female transports.	Total.																			
	Males.	Femls.			Males.	Females.*	Both sexes.	Males.	Females	Both sexes.	Males.	Females.	Both sexes.	Males.	Females.	Both sexes.	Males.	Females.	Both sexes.	Males.	Females.	Both sexes.	All males.
1824	106	24	..	130																			*
1825	130	64	..	194	167	61	228	3	..	3	..	..	..	18·0	..	13·1							
1826	205	21	..	226	339	92	431	11	4	15	..	..	..	32·4	43·4	34·5	..	..	..	32·4	43·4	34·5	
1827	142	35	..	177	474	105	579	9	2	11	5	3	8	18·9	19·0	19·0	10·5	28·5	13·8	29·5	47·6	32·8	
1828	177	74	..	251	503	120	623	13	3	16	7	13	20	25·8	25·0	25·6	13·9	108·3	32·1	39·7	13·3	57·7	
1829	61	29	..	90	533	136	669	13	5	18	19	20	39	24·3	36·7	26·9	35·6	147·0	56·8	60·0	18·3	85·2	7
1830	89	67	..	156	459	146	605	10	3	13	20	7	27	21·7	20·5	21·4	43·5	47·9	44·6	65·3	68·4	66·1	1
1831	149	39	..	188	394	149	543	6	4	10	6	9	15	15·2	26·8	18·2	15·2	60·4	27·6	30·4	87·1	47·6	1
1832	178	42	..	220	384	135	519	18	6	24	27	13	40	46·8	44·4	46·2	70·3	96·3	77·0	117·1	140·7	123·1	1
1833	198	48	..	246	440	141	581	8	2	10	15	12	27	18·1	14·1	17·2	34·0	85·1	46·4	52·2	99·2	63·6	
1834	168	31	..	199	520	117	637	15	1	16	13	15	28	28·8	8·5	25·1	25·0	128·2	43·9	53·8	136·7	69·0	
1835	87	29	..	116	467	97	564	5	..	5	27	15	42	10·7	..	8·8	58·2	154·6	74·4	68·9	..	68·9	4
1836	137	35	..	172	326	70	396	2	2	4	13	4	17	6·1	28·5	10·1	39·8	57·1	42·9	45·0	85·6	53·0	
1837	227	58	58	343	353	93	446	4	2	6	6	3	9	11·3	21·5	13·4	17·0	32·2	20·1	28·3	52·7	33·6	1
1838	80	40	144	264	311	134	445	8	2	10	9	9	18	25·7	14·9	22·4	28·9	67·1	40·4	54·6	82·0	62·9	10
1839	91	44	163	298	254	138	392	2	3	5	16	4	20	7·8	21·7	12·7	63·0	32·0	51·0	70·8	50·7	63·7	4
1840	432	100	520	1,052	385	237	622	5	1	6	7	6	13	12·9	4·2	9·6	18·4	29·5	20·9	31·1	29·9	30·6	
1841	126	40	10	176	498	194	692	11	2	13	7	7	14	22·0	10·3	18·7	14·0	36·0	20·2	36·1	46·8	39·0	
1842	131	49	3	183	456	160	616	16	4	20	16	2	18	35·0	24·8	32·2	35·1	12·5	29·4	70·1	37·3	61·6	
Whole period.	2,913	869	898	4,681	7263	2325	9588	159	46†	205	213	142‡	355	..	..	..	..	..	..	..	..	..	29
Annual average.	155·944	46·944	128·285	252·833	403·500	129·166	532·666	8·833	2·555	11·388	11·833	7·888	19·722	21·891	19·784	21·380	29·326	61·075	37·025	51·217	80·869	58·405	

Table II.—Showing the daily average number of criminal prisoners confined during the five years 1838 to 1842 inclusive, in each of 16 County Prisons, in which no Debtors, or a very inconsiderable number, were confined; and likewise the mortality of each of those Prisons during the same period.

	Average daily number of criminals.			Number of deaths.			Annual mortality per thousand prisoners.		
	Male.	Female.	Both sexes.	Male.	Female.	Both sexes.	Male.	Female.	Both sexes.
Salford House of Correction	525·	150·8	675·8	46	11	57	17·523	14·720	16·868
Wakefield House of Correction	516·6	82·2	598·8	78	5	83	30·778	12·141	27·722
Kirkdale House of Correction	338·6	50·6	389·2	31	2	33	18·310	7·905	16·957
Knutsford House of Correction	242·8	57·	299·8	27	2	29	22·240	7·017	19·346
Exeter County Gaol	66·	20·4	86·4	6	3	9	18·181	29·411	20·833
Exeter House of Correction	114·4	28·	142·4	13	2	15	22·709	14·857	20·224
Springfield County Gaol and House of Correction	181·	—	181·	13	—	13	14·364	—	14·364
Winchester House of Correction	149·8	15·	164·8	18	2	20	24·032	26·666	24·271
Coldbath-fields House of Correction	770·6	280·8	1,051·4	82	17	99	21·282	12·108	18·832
Brixton House of Correction	199·2	68·2	267·4	13	5	18	13·052	14·633	13·462
Guildford House of Correction	96·	18·2	114·2	10	—	10	20·833	—	17·513
Warwick House of Correction	206·2	33·2	239·4	29	2	31	32·953	12·422	25·876
Devizes House of Correction	162·2	19·8	182·	9	1	10	11·097	10·101	10·989
Preston House of Correction	185·4	39·4	224·8	4	2	6	3·358	1·779	5·338
Tothill-fields City Gaol.	188·2	114·6	302·8	33	7	40	35·069	12·216	26·418
Lancaster County Gaol	242·8	58·4	301·2	17	7	24	14·003	23·972	15·936
The 16 Prisons	4,184·8	1,036·6	5,221·4	429	68	497	20·454	13·111	19·033

TABLE III.—Showing the average number of criminal prisoners, and their mortality during the five years 1838 to 1842, in 20 County Prisons, into which more or less considerable numbers of Debtors were received.

	Average daily number of prisoners.			Number of deaths.			Annual mortality per thousand prisoners.		
	Male.	Female.	Both sexes.	Male.	Female.	Both sexes.	Male.	Female.	Both sexes.
Reading County Gaol and House of Correction	104·4	15·4	119·8	21	1	22	45·977	13·000	41·873
Maidstone County Gaol and House of Correction	312·6	56·2	367·8	35	4	39	22·392	14·456	21·207
Horsemonger Lane County Gaol	90·	14·8	104·8	5	3	8	11·062	40·540	15·019
Chester County Gaol	83·	17·2	100·2	8	4	12	19·277	46·511	23·483
Bodmin County Gaol and House of Correction	88·	23·2	111·2	6	1	7	13·636	8·620	12·589
Derby County Gaol and House of Correction	148·8	11·	159·8	7	3	10	9·408	54·545	12·516
Durham County Gaol and House of Correction	122·8	33·8	156·6	9	2	11	14·658	11·834	14·049
Gloucester County Gaol and Penitentiary	196·6	20·4	217·	10	3	13	11·729	29·412	11·981
Liverpool Borough Gaol	297·4	191·4	488·8	20	15	35	13·450	15·674	14·734
Newcastle Borough Gaol and House of Correction	62·8	22·	84·8	6	1	7	19·108	9·090	16·509
Shrewsbury County Gaol and House of Correction	102·2	18·8	121·	7	2	9	11·743	21·276	14·876
Stafford County Gaol and House of Correction	372·8	50·6	423·4	23	3	26	12·875	11·856	12·749
Warwick County Gaol	151·8	30·8	182·6	29	2	31	38·208	12·987	33·954
Worcester County Gaol and House of Correction	161·2	29·6	190·8	18	2	20	22·332	13·513	20·964
York County Gaol	54·4	6·8	61·2	9	5	14	32·573	147·059	45·751
Kingston-on-Hull Borough Gaol and County House of Correction.	67·8	19·2	87·	4	5	9	11·800	52·032	20·689
Dorchester County Gaol and House of Correction	90·8	11·6	102·4	10	1	11	22·026	17·276	21·584
Oxford County Gaol and House of Correction	109·8	14·4	124·2	14	3	17	25·500	41·666	27·326
Bristol City Gaol	98·2	23·8	122·	7	2	9	14·256	16·806	14·590
Aylesbury County Gaol and House of Correction	—	—	110·2	—	—	16	—	—	29·038
In all the 20 prisons	2,715·4	610·	3,435·6	248	63	326	17·898	20·000	18·622
In the 16 prisons named in *Table II.*	4,184·8	1,036·6	5,221·4	429	68	497	20·454	13·111	19·033
In all the 36 prisons	6,900·2	1,646·6	8,657·0	677	131	823	19·622	15·790	18·874

TABLE IV.—Number of Prisoners confined in Coldbath-fields Prison and Westminster Bridewell, and the number of Deaths in those Prisons, from the year 1825 to 1839 inclusive.

	Coldbath-fields County House of Correction.					Westminster Bridewell, or Tothill-fields City Gaol and House of Correction.				
Years.	Total number of prisoners during the year.	Greatest number of prisoners at one time.	Number of prisoners at Michaelmas.	Number of deaths during the year.	Number of deaths per 1,000 prisoners.	Total number of prisoners during the year.	Greatest number of prisoners at one time.	Number of prisoners at Michaelmas.	Number of deaths during the year.	Number of deaths per 1,000 prisoners.
1825	3,987	589	582	11	18·9	2,328	182	149	4	26·8
1826	4,922	723	642	15	23·3	2,829	186	180	3	16·6
1827	5,264	778	732	14	19·1	2,918	225	147	1	6·8
1828	5,884	886	860	10	11·6	3,742	211	131	2	15·2
1829	6,625	879	815	26	31·9	3,828	224	154	2	12·9
1830	7,485	909	859	13	15·1	4,604	271	160	2	12·5
1831	10,162	1,184	1,133	16	14·1	4,966	255	181	3	16·5
1832	12,543	1,325	1,184	64	53·2	3,857	226	108	3	27·7
1833	10,456	1,309	965	37	38·3	3,313	206	121		
1834	10,596	1,245	967	18	18·5	2,912	355	250	1	4·0
1835	7,598	968	825	26	31·5	5,604	464	295	6	20·3
1836	7,931	1,105	953	23	24·1	5,562	468	354	4	11·2
1837	8,760	1,240	1,238	42	33·9	5,771	438	435	16	36·7
1838	8,021	1,238	1,136	31	27·2	5,979	463	395	19	48·1
1839	8,689	1,228	1,142	12	10·5	6,378	476	340	8	23·5
Whole period	118,923	15,606	14,033	358	. .	64,591	4,650	3,400	74	
Annual average	7,928	1,040	935	24	24·7	4,306	310	226	5	20·0

The data for the construction of this table were obtained in the year 1840 from the Home Office. Deaths from Asiatic cholera are included.

TABLE V. — Showing the number of convicts on board the Hulks in England during each of the several years, 1825 to 1839, with the number of Deaths in each year.

Years.	On board 1st January.	Received during the year.	Total number during the year.	Average number during the years.	Deaths in each year.	Deaths per 1,000 of average number of convicts.
1825	3,230	2,184	5,414	3,438	108	31·4
1826	3,159	2,776	5,935	3,610	108	29·9
1827	3,994	3,748	7,742	4,362	166	38·0
1828	4,446	3,464	7,910	4,414	125	28·3
1829	4,185	4,230	8,415	4,446	158	35·5
1830	4,250	4,087	8,337	4,142	126	30·4
1831	4,071	4,551	8,622	4,152	133	32·0
1832	4,139	4,627	8,766	4,211	262*	62·2
1833	3,898	4,556	8,454	3,530	164	46·4
1834	3,060	4,374	7,434	3,015	142	47·0
1835	2,556	4,063	6,619	2,487	137	55·0
1836	2,202	4,102	6,304	2,244	117	52·1
1837	1,935	3,970	5,905	1,971	120	60·8
1838	1,789	4,138	5,927	2,130	113	53·0
1839	2,047	3,841	5,888	2,697	111	41·1
Aggregate numbers during the entire period 1825 to 1839 inclusive.	48,961	58,711	107,672	50,849	2,090	41·10
Annual average of the entire period, 110 deaths from cholera being excluded from the calculation.	3,264	3,914	7,178	3,389	132	38·938

* 110 deaths from cholera in this year.

TABLE VI.—Showing the number of prisoners confined in the Penitentiary of *Geneva*, and the mortality suffered by them during the 16 years, 1826 to 1841 inclusive.

Years.	Number received.	Average daily number.	Deaths.	Ratio per 1,000 of deaths to the average number of prisoners.
1826	258	36·30	2	55·1
1827		47·69		
1828		49·36		
1829		49·73	2	40·2
1830		59·30	1	16·8
1831		56·23	1	17·6
1832		54·66	1	18·2
1833	64	64·20	1	15·5
1834		62·44	3	49·4
1835	75	60·85	1	16·4
1836		60 82	2	32·8
1837		60·98	3	49·1
Period of 12 years; aggregate numbers	397	662·56	17	
1838	—	62·14	2	32·185
1839	—	56·41	2	35·454
1840	—	65·98	2	30·312
1841	—	62·69	1	15·951
Whole period of 16 years; aggregate numbers	—	909·78	24	
Annual average.	—	56·911	1·5	26·380

TABLE VII.—Showing the number of prisoners confined in the Penitentiary of Lausanne, and the mortality suffered by them during the period of 15¼ years, from October 1, 1826 to December 31, 1841.

Years.	Number received.	Average daily number.	Deaths.	Ratio per 1,000 of deaths to the average number of prisoners.
Year ending Sept. 30, 1827	42	78	3	38·461
1828	37	84	4	47·619
1829	41	82	3	36·585
1830	41	75	6	80·000
1831	47	74	2	27·027
1832	73	77	1	13·000
15 months ending Dec. 31, 1833	111	93	5	43·103
Year ending Dec. 31, 1834	58	91	4	43·956
1835	84	83	2	24·096
1836	72	84	1	11·905
1837	71	82	6	73·170
1838	86	94	3	31·915
1839	78	106	5	47·169
1840	88	113	3	26·548
1841	76	105	4	38·095
Whole period; aggregate numbers	1,005	1,344	52	
Annual average	66	88·144	3·409	38·690

TABLE VIII.—Showing the number of prisoners of the White and Dark Races who were confined in the Eastern Penitentiary of Pennsylvania during the fourteen years, 1830 to 1843 inclusive, and the mortality amongst each of these classes of Prisoners.

Years.	White Prisoners.				Coloured Prisoners.				All Prisoners.			
	Received during the year.	Average daily number.	Deaths.	Ratio per 1,000 of deaths to the average number of prisoners.	Received during the year.	Average daily number.	Deaths.	Ratio per 1,000 of deaths to the average number of prisoners.	Received during the year.	Average daily number.	Deaths.	Ratio per 1,000 of deaths to the average number of prisoners.
1830	107	21·81	1	45·80	35	9·19	—	—	58	31	1	32·2
1831		47·75	2	41·88		19·25	2	103·89	50	67	4	59·7
1832		69·42	1	14·40		21·58	3	139·01	34	91	4	43·9
1833	55	89·30	1	11·12	22	33·70	—	—	77	123	1	8·0
1834	76	123·58	1	8·09	42	59·42	4	67·31	118	183	5	27·3
1835	121	157·74	2	12·67	96	108·26	5	46·17	217	286	7	26·3
1836	84	202	2	9·90	59	148	10	67·56	143	360	12	33·3
1837	101	233	7	30·42	60	154	10	65·93	161	386	17	44·0
1838	115	241	7	29·04	63	161	19	118·01	178	402	26	63·1
1839	99	245	2	81·59	80	173	9	53·02	179	418	11	26·3
1840	88	232	9	38·79	51	162	13	80·24	139	394	22	55·83
1841	83	201	4	19·40	43	134	13	97·01	126	335	17	50·74
1842	102	212	3	14·15	40	119	6	50·42	142	331	9	27·19
1843	113	235	5	21·27	43	124	6	48·38	156	359	11	30·64
Whole period	1,144	2,310·60	47	—	634	1,426·40	100	—	1,778	3,737	147	39·33
Annual average	81·71	165·04	3·36	20·34	45·29	101·88	7·14	70·10	127	266·92	10·5	39·336

TABLE IX.—Showing the number of prisoners and the mortality in the Auburn State Prison (New York) during the sixteen years, 1825 to 1840 inclusive.

Years.	Number received during the year: White prisoners.	Coloured prisoners.	All prisoners.	Number on the list of Inmates.	Deaths.	Ratio per thousand of deaths to the average number of prisoners.
1825	146	8	154	427	6	14·051
1826	123	10	133	427	7	16·394
1827	172	18	190	525	9	17·142
1828	156	18	174	571	9	15·761
1829	150	20	170	639	6	7·824
1830	102	12	114	620	18	29·032
1831	154	20	174	647	15	21·483
1832	157	35	192	683	12	17·569
1833	176	17	193	679	11	16·200
1834	161	27	188	679	11	16·200
1835	202	26	228	654	10	15·290
1836	160	23	183	648	18	27·777
1837	227	30	257	705	19	26·950
1838	165	23	218	616	15	24·350
1839	228	16	244	670	10	14·925
1840	—	—	225	695	14	20·143
Whole period	2,499*	313*	3,040†	9,885	190	19·220
Annual average.	166·60	20·866	190·	617·86	12·75	19·019

* During fifteen years. † During sixteen years.

Table X.—Showing the number of prisoners and the mortality in the Maryland Penitentiary (at Baltimore) during the twelve years, 1830 to 1840, and 1843.

Years.	Number received during the year.			Average daily number.	Deaths.	Ratio per 1,000 of deaths to the average number of prisoners.
	White prisoners.	Coloured prisoners.	All prisoners.			
1830	59	58	117	363	13	35·812
1831	44	61	105	370	24	64·864
1832	46	69	115	269	22	81·784
1833	50	70	120	356	16	44·382
1834	47	76	123	369	10	27·100
1835	84	60	144	390	11	28·205
1836	45	59	104	401	6	14·962
1837	55	73	128	391	13	33·248
1838	46	45	91	370	11	29·729
1839	41	46	87	340	11	32·352
1840	44	60	104	328	14	42·073
1843	—	—	—	387	21	54·263
Whole period	561	677	1,238	4,334	172	
Annual average	52·33	60·77	113·11	361·16	14·33	39·686
Annual average mortality when 17 deaths caused by Asiatic cholera are excluded from the calculation					12·916	35·763

TABLE XI.—Showing the number of prisoners and the mortality in the State Prison of Charlestown (Massachusetts) during the 12 years, 1829 to 1840 inclusive.

Years.	Received during the year.	Number in prison on the 30th September in each year.			Deaths.	Ratio per 1,000 of deaths, to the number in prison.
		White prisoners.	Coloured prisoners.	All prisoners.		
1829	79	230	32	262	6	22·90
1830	115	252	38	290	5	17·241
1831	71	215	41	256	7	27·343
1832	76	192	35	227	11	48·458
1833	119	219	31	250	6	24·0
1834	119	243	34	277	4	14·440
1835	116	247	32	279	3	10·752
1836	97	250	28	278	4	14·388
1837	99	267	24	291	5	17·113
1838	114	277	26	303	7	23·102
1839	104	291	27	318	5	15·723
1840	103	285	33	318	2	6·289
Whole period.	1,212	2,968	381	3,349	65	
Annual average.	·101	247·33	31·75	279·08	5·41	19·408

TABLE XII.—Showing the number of prisoners and the mortality in the State Prison of Wethersfield (Connecticut) during the 13 years, 1830 to 1839 inclusive, and 1842 to 1844 inclusive.

Years.	Received during the year.	Number in prison on the 31st March in each year.			Deaths.	Ratio per 1,000 of deaths to the number of prisoners.
		White prisoners.	Coloured prisoners.	All prisoners.		
1830	73	128	39	167	4	23·952
1831	55	..	..	182	4	21·978
1832	65	156	36	192	2	10·411
1833	52	..	..	186	3	16·129
1834	54	137	52	189	1	5·291
1835	75	157	50	207	4	19·323
1836	66	153	48	201	8	39·800
1837	57	165	42	207	1	4·831
1838	57	142	49	191	4	20·942
1839	50	135	48	183	9	49·180
1842	..	159	52	211	10	45·300
1843	..	155	48	203	8	39·408
1844	..	146	49	195	10	51·282
Aggregate.	604	1,633	503	2,514	68	
Annual Average.	60·40	148·454*	45·727*	193·38†	5·23	27·048

* During 11 years. † During 13 years.

TABLE XIII.—Showing the number of prisoners and the mortality in the State Prison of Sing Sing (New York) during the 13 years, 1828 to 1840 inclusive.

Years.	Number received during the year.	Number discharged, pardoned, escaped, removed, or died.	Number in prison on the 30th September in each year.	Deaths.	Ratio per 1,000 of deaths to the number in prison.
1828	146	151	512	16	31·250
1829	168	90	590	14	23·728
1830	363	143	810	16	19·753
1831	359	189	980	28	28·571
1832	289	437	832	153	183·894
1833	219	240	811	25	30·826
1834	258	215	843	18	21·352
1835	213	260	796	31	38·944
1836	182	252	726	11	15·151
1837	291	236	781	20	25·608
1838	323	262	842	33	39·192
1839	209	246	805	34	42·236
1840	254	232	827	17	20·556
Whole period.	3,274	2,953	10,155	416	
Annual average.	251·846	227·149	781·153	32·0	40·960
Annual average mortality, where 103 deaths caused by Asiatic cholera are excluded from the calculation.				24·07	30·822

TABLE XIV.—Showing the proportion borne by the prisoners of the dark or coloured races to the whole number of prisoners in the Sing Sing State Prison at different periods.

	Number of prisoners received.						Discharged during	In prison at one time in
Years.	1828	1829	1830	1831	1832	Whole period.	1839	1836
White prisoners.	123	136	332	289	244	1,124	194	616
Coloured prisoners.	23	32	31	70	45	201	52	201
All prisoners.	146	168	363	359	289	1,325	246	817
Proportion per 100 of coloured prisoners to the whole number.	15·75	19·04	8·54	19·49	15·53	15·17	21·13	24·60

Table XV.—Showing the total number of prisoners received into 15 county prisons during five years, the average number of prisoners constantly in each of these prisons, and the average term of confinement in each prison.

	Total numbers of prisoners received in the 5 years, 1838 to 1842 inclusive.	Aggregate of average daily numbers of prisoners in the 5 years.	Average term of confinement of the prisoners in each prison.
Knutsford House of Correction.	7,858	1,499	69
Exeter County Gaol.	3,449	432	45
Exeter House of Correction.	3,908	712	66
Springfield County Gaol and House of Correction.	5,225	905	63
Winchester County House of Correction.	4,306	824	69
Preston County House of Correction.	8,223	1,170	58
Kirkdale County House of Correction.	11,034	1,946	64
Salford County House of Correction.	29,035	3,379	42
Coldbath-fields County House of Correction.	47,778	5,257	40
Tothill-fields City Gaol and House of Correction.	26,994	1,529	20
Guildford County Gaol and House of Correction.	2,886	571	72
Brixton County House of Correction.	13,055	1,337	37
Warwick County House of Correction.	5,933	1,198	73
Devizes County House of Correction.	4,634	910	71
Wakefield County House of Correction.	17,665	2,994	62
The 15 prisons.	191,983	24,663	46

TABLE XVI.—Showing the number of military offenders who were confined in the Millbank Penitentiary between the 18th November 1837 and the end of 1842, the number who died, and the number who were pardoned on medical grounds.

Years.	Number received.	Average daily number.	Deaths.	Pardons on medical grounds.	Deaths and pardons.	Deaths per thousand prisoners.	Pardons per thousand prisoners.	Deaths and pardons per thousand prisoners.
1837	55							
1838	611	92						
1839	697	125						
1840	760	140	1	1	2	7·142	7·142	14·287
1841	728	145	2	2	4	13·793	13·793	27·586
1842	741	130	—	1	1	0·000	7·692	7·692
Aggregate for the whole period of 5 years.	3,592	652	3	4	7			
Annual average.	707·4	130·4	·6	·8	1·4	4·601	.6·135	10·736

TABLE XVII.—Showing the diseases which caused death, or formed the grounds of pardon amongst the military offenders in the Millbank Penitiary during five years.

	Deaths.					Pardons.					Aggregate deaths and pardons due to each disease.	Ratio per 1,000 of deaths and pardons from each disease to the average number of prisoners.
Years.	1838	1839	1840	1841	1842	1838	1839	1840	1841	1842		
Erysipelas	..	..	1	..	..	..	..	..	..	..	1	1·533
Chronic dysentery	..	..	..	..	..	..	..	..	..	1	1	1·533
Weakness of mind	..	..	..	..	..	..	..	..	1	..	1	1·533
Consumption	..	..	..	1	..	..	..	1	1	..	3	4·601
Bronchitis	..	..	..	1	..	..	..	..	..	..	1	1·533
All causes	..	..	1	2	..	..	..	1	2	1	7	10·736

Table XVIII.—Showing the number of deaths, and the annual rate of criminals in the Millbank Penitentiary, from the

Years.		1825	1826	1827	1828	1829	1830	1831	1832
Average number of prisoners.		228	431	579	623	669	605	543	519
Fevers.	Continued fever.	1	2	2	4	4	2	1	3
	Ague.	..	..	..	..	..	..	1	..
Asiatic cholera.		..	..	..	..	..	..	..	17
Other epidemic diseases.	Smallpox.	..	..	..	..	..	..	..	..
	Erysipelas.	..	2	..	1	..	..	..	..
	Influenza.	..	..	..	..	..	..	..	..
Consumption.	Consumption.	..	5	7	8	7	10	4	..
	Hæmoptysis.	..	..	..	..	..	..	..	..
Other tubercular diseases.	Tubercular arachnitis.	..	..	..	..	..	..	..	..
	Tubercular pleuritis.	..	..	..	..	..	..	..	..
	Tubercular peritonitis.	..	..	..	..	..	..	..	..
	Mesenteric disease.	..	..	..	..	..	..	..	..
	External glandular scrofula.	..	2	2	..	..	..	..	..
Diseases of the brain and nervous system.	Phrenitis.	..	..	..	1	..	..	..	..
	Acute Hydrocephalus.	..	..	..	..	1	..	..	..
	Apoplexy.	..	..	..	..	..	..	1	..
	Epilepsy.	..	..	..	..	1	..	..	..
	Hydrophobia.	..	..	..	..	..	..	..	..
	Insanity.	..	..	..	..	1	..	..	..
Diseased heart.		..	1	..	..	..	..	..	..
Diseases of the respiratory organs, exclusive of consumption.	Asthma.	..	..	..	..	..	..	..	..
	Pneumonia.	..	..	..	..	..	..	..	..
	Pleuritis.	..	..	..	..	..	..	..	..
	Hydrothorax with diseased lungs	..	..	..	..	..	..	..	..
	Inflammation of chest.	..	..	..	..	1	..	..	..
Diseased liver.		..	..	..	..	1	..	..	..
Bowel complaints.	Dysentery.	..	..	..	..	..	..	..	..
	Tetanic cramps, with dysentery	..	..	..	..	..	..	..	..
	Common cholera.	1	..	..	..	..	..	..	..
	Diarrhœa.	..	..	..	..	..	..	1	..
	Enteritis.	..	..	..	..	..	1	..	..
	Ulceration of bowels.	..	..	..	..	..	..	..	..
Other diseases of the abdomen.	Obstruction of bowels.	..	..	..	1	..	..	..	..
	Hernia.	..	..	..	..	..	..	..	..
	Peritonitis.	..	1	..	..	..	..	..	..
Other diseases not classified.	Dropsy, with asthma.	..	1	..	..	1	..	..	..
	Acute rheumatism.	..	1	..	..	..	..	..	..
	Abscess.	..	..	..	..	..	..	..	..
Diseases of undefined nature.	Visceral disease.	..	..	..	1	1	..	..	3
	Debility.	..	..	..	..	..	..	1	..
Suicide.		1	..	..	..	..	..	1	1
All causes.		3	15	11	16	18	13	10	24

mortality caused by different diseases and classes of diseases amongst the 1st January 1825 to 31st December 1842.

1833	1834	1835	1836	1837	1838	1839	1840	1841	1842	Whole period.	Annual ratio of deaths per thousand prisoners from each disease.	Total number of deaths from each class of diseases.	Annual ratio of deaths per thousand prisoners from each class of diseases.
581	637	564	396	446	445	392	622	692	616	9,588			
2	4	4	1	..	1	..	2	1	..	34	3·546	35	3·650
..	..	..	..	..	..	..	..	..	..	1	·104		
5	9	..	..	..	..	..	..	..	..	31	3·233	31	3·233
..	..	..	..	..	..	..	..	1	..	1	·104		
..	..	..	..	1	..	..	..	..	..	4	·417	8	·834
..	1	..	..	2	..	..	..	..	..	3	·313		
2	1	..	2	2	3	4	4	7	6	72	7·509	73	7·613
1	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	1	1	·104		
..	..	..	..	..	..	..	..	..	1	1	·104		
..	..	..	..	..	..	..	..	..	1	1	·104	8	·834
..	..	..	..	..	1	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	..	4	·417		
..	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	1	2	·208	7	·730
..	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	1	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	..	1	·104	1	·104
..	..	1	..	..	..	..	..	..	..	1	·104		
..	1	..	..	..	..	..	..	..	1	2	·208		
..	..	..	..	..	..	..	..	1	..	1	·104	6	·625
..	..	..	..	..	1	..	..	..	..	1	·104		
..	..	..	..	..	.	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	.	..	1	·104	1	1·04
..	..	..	..	..	..	..	..	2	8	10	1·043		
..	..	..	..	..	..	..	..	..	1	1	·104		
..	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	2	..	..	1	..	4	·417	18	1·877
..	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	..	1	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	1	..	..	..	..	..	..	1	·104	3	·312
..	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	..	2	·208		
..	..	..	..	..	..	..	..	..	..	1	·104	4	·417
..	..	..	..	..	1	..	..	..	..	1	·104		
..	..	..	..	1	..	..	..	..	..	6	·625	7	·730
..	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	..	3	·313	3	·313
10	*16*	*5*	*4*	*6*	*10*	*5*	*6*	*13*	*20*	205	21·380	205	21·380

TABLE XIX.—Showing the total number, and the annual ratio, of par- the criminal prisoners in the Millbank Penitentiary, from the

Years.		1825	1826	1827	1828	1829	1830	1831	1832
Average number of prisoners.		228	431	579	623	669	605	543	519
	Ague.	..	..	..	..	..	..	..	..
Consumption.	Consumption.	..	..	1	5	3	9	5	8
	Hæmoptysis.	..	..	..	..	1	..	..	..
	Scrofula.	..	..	5	3	6	9	4	10
Diseases of the brain and nervous system.	Chronic hydrocephalus.	..	..	..	..	..	..	..	..
	Hemiplegia.	..	..	..	..	..	..	..	..
	Paraplegia.	..	..	..	..	1	..	..	..
	Paralysis.	..	..	..	..	..	..	1	..
	Chorea.	..	..	1	..	..	..	..	..
	Epilepsy.	..	..	1	..	4	1	..	1
	Hysteria.	..	..	..	..	..	..	1	1
	Unsound or weak mind.	..	..	..	1	..	..	..	..
	Neuralgia.	..	..	..	..	..	..	..	..
	Disease of the head.	..	..	..	..	..	..	..	..
	Pain in the head.	..	..	..	..	..	..	..	1
Diseases of the heart.	Diseased heart.	..	..	..	2	1	1	..	1
	Pericarditis, with debility.	..	..	..	..	..	..	..	..
Diseases of the respiratory organs, exclusive of consumption.	Asthma.	..	..	..	1	1	1	..	..
	Aphonia.	..	..	..	..	..	..	..	..
	Diseased lungs.	..	..	..	1	1	..	..	1
	Chest affection, with debility.	..	..	..	..	1	..	..	..
	Spasmodic affection of chest.	..	..	..	..	1	..	..	..
	Diseased liver.	..	..	..	..	1	..	..	..
Bowel complaints.	Dysentery.	..	..	..	..	..	1	..	..
	Diarrhœa.	..	..	..	..	..	..	..	2
Other diseases of the abdomen.	Hæmatemesis.	..	..	..	..	1	..	1	..
	Hæmorrhage from bowels.	..	..	..	..	..	..	..	..
	Stricture of intestine.	..	..	..	1	..	..	..	..
	Diseased rectum.	..	..	..	..	..	..	..	..
	Hernia.	..	..	..	..	..	..	..	..
	Disorder and debility of dig. org.	..	..	..	..	..	..	..	..
Diseases of the urinary and generative organs.	Diseased bladder.	..	..	..	..	..	..	..	..
	Cancer of uterus.	..	..	..	..	..	..	1	..
	Prolapsus of uterus.	..	..	..	..	..	1	..	..
	Diseased uterus.	..	..	..	1	..	..	..	1
	Menorrhagia.	..	..	..	..	..	..	..	..
	Uterine complaint.	..	..	..	..	..	..	..	1
Other diseases not classified.	Chronic rheumatism.	..	..	..	1	1	..	..	1
	Gout.	..	..	..	..	..	..	..	..
	Diseased joints.	..	..	..	..	1	..	..	2
	Diseased hand, with debility.	..	..	..	..	..	..	..	..
	Stone in the bladder.	..	..	..	..	..	..	..	..
	Abscess.	..	..	..	..	..	1	..	..
	Fistula in ano.	..	..	..	..	1	..	..	..
	Necrosis.	..	..	..	..	..	..	..	..
	Ulcers of legs.	..	..	..	..	1	..	..	..
	External tumours.	..	..	..	..	..	..	..	..
	Internal abdominal tumours.	..	..	..	..	..	..	..	..
	Disease of spine, with debility.	..	..	..	..	..	..	..	1
Diseases of *undefined nature.*	Visceral disease.	..	..	..	..	3	1	1	3
	General ill health.	..	..	..	2	7	..	1	4
	Debility.	..	..	..	1	1	2	..	1
	Declining health.	..	..	..	1	2	..	..	1
All causes.					20	39	27	15	40

dons granted on account of different diseases and classes of diseases, to 1st January 825 to the 31st December 1842.

1833	1834	1835	1836	1837	1838	1839	1840	1841	1842	Whole period.	Annual ratio of pardons per thousand prisoners, on account of each disease.	Total number of pardons on account of each class of diseases.	Annual ratio of pardons per thousand on account of each class of diseases.
581	637	564	396	446	445	392	622	692	616	9,588			
..	1	..	..	..	..	1	..	..	..	2	·208	2	·208
3	4	13	3	1	2	8	8	7	9	89	9 282	90	9·387
..	..	..	..	..	..	..	..	..	..	1	·104		
3	6	13	4	3	7	1	2	1	1	78	8·135	78	8·135
1	..	..	..	..	..	..	..	..	..	1	·104		
1	1	..	..	..	..	..	..	..	..	2	·208		
..	..	..	..	..	..	..	..	..	..	1	·104		
..	..	1	..	..	..	..	..	..	..	2	·208		
..	..	..	..	..	..	..	..	..	..	1	·104		
3	..	3	..	..	..	..	..	1	..	14	1·460	31	3·233
..	..	..	..	..	..	..	..	..	..	2	·208		
..	..	..	2	..	..	1	..	..	1	5	·521		
..	..	..	..	1	..	..	..	..	..	1	·104		
..	1	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	..	1	·104		
2	..	..	..	..	..	..	..	..	..	7	·730	8	·834
..	..	..	..	..	..	..	..	..	1	1	·104		
..	..	1	1	..	..	1	..	..	..	6	·625		
..	..	1	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	..	3	·313	14	1·460
1	..	1	..	..	..	..	..	..	..	3	·313		
..	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	..	1	..	..	..	2	·208	2	·208
..	..	..	..	..	..	..	..	..	1	2	·208	14	1·460
2	2	..	..	..	2	1	2	1	..	12	1·251		
..	..	..	..	..	..	..	..	..	..	2	·208		
..	..	..	..	..	..	1	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	..	1	·104	10	1 043
1	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	1	..	..	..	..	..	..	1	·104		
..	..	..	..	2	..	2	..	..	..	4	·417		
1	1	..	..	..	..	..	..	..	..	2	·208		
..	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	..	1	·104	11	1·147
..	2	..	1	..	..	..	..	..	..	5	·521		
..	..	..	..	1	..	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	..	..	1	·104		
1	..	3	1	..	..	..	..	..	..	8	·834		
..	1	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	..	..	..	1	..	4	·417		
..	..	..	..	..	..	..	..	..	1	1	·104		
..	..	..	..	..	..	..	..	..	1	1	·104		
..	..	..	..	..	..	..	..	..	..	1	·104		
..	..	..	..	..	1	..	..	..	..	2	·208	27	2·816
1	..	..	..	..	..	..	..	..	..	1	·104		
..	..	1	..	..	..	..	..	..	1	3	·313		
..	..	..	..	..	..	..	1	..	..	1	·104		
..	..	1	..	..	..	..	..	1	..	2	·208		
..	..	..	1	..	..	..	..	..	..	2	.208		
2	..	1	1	1	4	..	..	..	..	17	1·772		
4	8	1	1	..	..	1	..	..	..	29	3·025	68	7·091
1	1	2	1	..	2	..	..	2	2	16	1·668		
..	..	..	..	..	..	2	..	..	..	6	·625		
27	*28*	*42*	*17*	*9*	*18*	*20*	*13*	*14*	*18*	355	37·025	355	37·025

TABLE XX.—*Showing the mortality caused by different diseases amongst the Criminal Prisoners in 16 County Prisons into which few or no Debtors were received.*

		Wakefield House of Correction.	Salford House of Correction	Coldbathfields House of Correction.	Knutsford House of Correction.	Devizes House of Correction.	Kirkdale County Gaol & House of Correction.	Springfield County Gaol & House of Correction.	Warwick House of Correction.	Brixton House of Correction.	Guildford House of Correction.	Winchester House of Correction.	Exeter House of Correction.	Exeter County Gaol.	Lancaster County Gaol.	Tothillfields City Gaol and H. of Correction.	Preston House of Correction.	Aggregate number of prisoners and of deaths.	Mortality per thousand prisonersfrom each disease.	Deaths from each class of diseases	Mortality per thousand prisonersfrom each class of diseases.
Years.		9 yrs. 1835 to 43	5 yrs. 37—40 & 42	3 yrs. 1839 to 1841	7 yrs. 1834 to 1840	12 yrs. 1829 to 1840	5 yrs. 37,38& 40—42	3 yrs. 1839 to 1841	1 year, 1839	2 yrs. 1839 & 40	2 yrs. 1839 & 40	1 year, 1838	1 year, 1839	1 year, 1839	19 yrs. 1825 to 1843	3 yrs. 1839 to 1841	4 yrs. 37, 38, 40 &42				
Aggregate of the annual averages of prisoners.		5,050	3,225	3,153	1,725	2,096	2,046	507	234	534	223	167	136	87	5,011	860	874	25,928			
Total number of deaths.		135	56	53	34	32	37	7	7	4	4	3	1	2	102	17	5	499	15·388	499	15·388
Fevers.	Typhus fever.	28	6	2	3	9	6	1	1	...	...	...	...	...	4	2	...	62	2·391	66	2·545
	Severe fever and diarrhœa.	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Gastric or bilious fever.	...	...	1	...	...	...	...	...	...	1	...	...	...	1	...	...	3	·116		
Epid. dis. not classified.	Erysipelas.	5	...	...	...	...	...	...	...	...	...	...	...	...	2	...	...	7	·269	9	·346
	Influenza.	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	·077		
Consumption.	Consumption.	18	13	...	10	7	9	...	2	...	2	...	...	1	32	1	2	97	3·741	102	3·933
	Hæmoptysis.	2	...	...	1	...	...	...	...	...	...	1	...	...	...	...	1	5	·192		
Other tubercular diseases.	Scrofula.	1	...	...	...	...	...	...	...	...	...	...	1	...	4	...	...	6	·231	7	·269
	Mesenteric disease.	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
Diseases of the brain and nervous system.	Phrenitis or brain fever.	...	1	...	...	...	1	...	...	...	...	...	...	...	...	...	...	2	·077	53	2·043
	Effusion on or congest. of brain.	6	1	...	...	...	...	...	...	...	...	...	...	...	4	...	...	11	·424		
	Hydrocephalus.	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Abscess of the brain.	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Apoplexy.	1	4	2	3	4	1	...	...	1	...	...	...	...	...	1	...	17	·655		
	Epilepsy.	...	2	...	...	...	...	...	...	...	...	...	...	...	...	2	...	4	·153		
	Hydrophobia.	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Tetanus.	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Paralysis.	1	...	5	...	2	...	...	...	...	...	...	...	...	3	...	...	11	·424		
	Delirium tremens.	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	1	·038		
	Diseased brain.	...	2	...	1	...	...	...	...	...	...	...	...	...	...	...	...	3	·115		
Diseases of the heart.	Diseased heart with dropsy.	1	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	2	·077	4	·153
	Angina Pectoris.	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Hydropericardium.	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
Diseases of the respiratory organs, exclusive of consumption.	Asthma.	...	4	8	...	...	2	...	3	...	...	...	...	...	3	...	...	20	·772	101	3·895
	Chronic Laryngitis.	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Bronchitis.	12	...	4	...	...	1	...	...	...	...	...	...	...	1	...	...	18	·694		
	Pneumonia.	4	3	...	2	1	...	...	...	...	1	1	...	...	...	...	...	12	·462		
	Inflammation of chest.	1	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	2	·077		
	Pleurisy.	3	...	3	...	...	...	...	...	...	...	...	...	...	...	1	...	7	·269		
	Hydrothorax.	6	4	...	1	...	3	...	...	...	...	...	...	...	6	1	...	21	·810		
	Congestion of the lungs.	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Diseased lungs.	1	9	3	...	...	...	1	...	...	...	...	...	...	1	...	...	15	·578		
	Affection of the chest.	...	...	4	...	...	...	...	...	...	...	...	...	...	...	...	...	4	·153		

Hepatic diseases.	Hepatitis.	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038	4	·153
	Diseased liver.	...	...	...	...	...	1	...	...	...	...	...	...	...	...	1	...	...	2	·077		
	Jaundice.	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
Bowel complaints.	Dysentery.	4	...	...	3	3	...	...	...	...	...	...	...	...	...	10	...	...	20	·772	54	2·081
	Diarrhœa.	6	...	7	...	...	...	...	...	...	...	...	...	...	...	2	3	...	18	·694		
	Cholera.	1	...	...	...	...	1	...	...	...	...	...	...	...	...	3	...	...	5	·192		
	Enteritis.	3	...	...	...	1	...	1	...	...	...	...	...	...	1	...	...	...	6	·231		
	Ulceration of the bowels.	2	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	3	·115		
	Intestinal disease.	...	2	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	·077		
Other diseases of the abdomen.	Gastritis.	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038	11	·424
	Spasm of the stomach.	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Diseased stomach.	...	...	...	2	...	...	...	...	...	...	...	...	...	...	1	...	1	4	·153		
	Colic.	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Peritonitis.	1	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	·077		
	Diseased rectum.	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Hernia.	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	1	·038		
Diabetes.		...	...	...	1	...	...	...	...	...	...	...	...	...	...	1	...	...	2	·077	2	·077
Cancer uterus.		...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038	1	·038
Other diseases not classified.	Dropsy.	5	...	2	...	2	5	...	1	1	...	...	...	...	...	5	2	...	23	·887	32	1·234
	Rheumatic fever.	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	1	·038		
	Abscess.	...	...	...	1	...	...	...	...	...	...	1	...	...	...	1	...	...	3	·115		
	Mortification of legs and feet.	...	...	2	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	·077		
	Phagedœna of leg, with debility.	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Diseased joints.	1	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	2	·077		
Diseases of undefined nature.	Debility.	5	1	4	...	...	5	2	...	2	...	...	...	...	...	7	...	...	26	1·003	48	1·851
	Do. from dis. of heart & stom.	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Atrophy and marasmus.	3	...	...	...	...	...	...	...	...	...	...	...	...	...	5	...	...	8	·115		
	Cachexia.	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	...	...	2	·077		
	Diseased liver and lungs.	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Low fever with fits.	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Broken constitution.	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		
	Natural death.	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	1	·038		
	Old age.	...	...	...	...	1	2	1	...	...	...	...	...	...	...	...	...	...	4	·153		
	Found dead in bed.	...	...	2	...	...	...	...	...	...	...	...	...	...	...	1	...	...	3	·115		
Violent deaths.	Rupt. intest. from ext. violence.	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	1	·038	5	·192
	Suicide.	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	3	·115		
	Accidental suffocation.	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	·038		

TABLE XXI.—Showing the mortality caused by different diseases amongst the prisoners in 17 county prisons (the debtors who were received into these prisons not being included).

Prisons.		Reading County Gaol and House of Correction.	Maidstone County Gaol and House of Correction.	Horsemonger Lane County Gaol.	Chester County Gaol.	Bodmin County Gaol and House of Correction.	Derby County Gaol and House of Correction.	Gloucester County Gaol and Penitentiary.	Liverpool Borough Gaol.	Stafford County Gaol and House of Correction.	Warwick County Gaol.	Worcester County Gaol and House of Correction.	York County Gaol.	Hull Town Gaol and County House of Correction.	Dorchester County Gaol and House of Correction.	Oxford County Gaol and House of Correction.	Bristol Common Gaol.	Aylesbury County Gaol and House of Correction.	Aggregate number of prisoners and of deaths.	Mortality per thousand from each disease.	Deaths from each class of diseases.	Mortality per thousand from each class of diseases.
Periods.		3 years 1839 to 1841.	2 years 1839 and 40.	2 years 1839 and 40.	4 years 1838, 40, 1842, and 43.	2 years 1838 and 39.	6 years 1837, 38, 1840 to 43.	3 years 1837, 39, and 41.	5 years 1837, 38, 1840 to 42.	1 year 1839.	1 year 1839.	1 year 1839.	8 years 1833 to 38, 1840 and 41.	2 years 1840 and 41.	2 years 1839 and 40.	3 years 1838 to 40.	3 years 1837, 38, and 40.	2 years 1840 and 41.				
Aggregate of the annual averages of prisoners.		379	705	202	471	218	997	602	2347	389	153	178	397	169	250	365	325	280	8427			
Total number of deaths.		17	9	2	10	3	13	9	41	11	4	2	27	3	2	7	18	3	181	21·478	181	21·478
Fever.		..	1	..	3	..	1	..	5	1	..	1	..	1	..	1	2	..	16	1·898	16	1·898
Epid. dis. not classified	Erysipelas.	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	2	·237	5	·593
	Smallpox.	..	..	..	..	..	1	1	..	..	..	..	1	..	..	..	..	..	3	·356		
Consumption.	Consumption.	5	1	1	4	1	2	3	16	3	1	..	7	..	1	1	4	..	50	5·933	54	6·407
	Hæmoptysis.	1	2	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	4	·474		
Scrofula.		..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	·118	1	·118
Diseases of the brain and nervous system.	Brain fever.	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	·118	25	2·964
	Effusion on the brain	..	..	..	..	..	..	..	1	..	..	..	..	..	..	1	..	..	2	·237		
	Diseased brain.	..	1	..	..	..	..	1	1	1	..	..	..	..	..	1	..	..	5	·593		
	Apoplexy.	1	1	..	..	1	..	..	2	1	1	..	1	..	..	..	3	..	11	1·305		
	Epilepsy.	..	..	..	..	..	1	..	1	1	..	..	..	..	..	..	..	..	3	·356		
	Tetanus.	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	·118		
	Depress. of the nerv. sys.	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	·118		
	Paralysis.	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1	.118		

Diseased heart.		..	..	..	..	..	1	1	2	1	..	..	..	..	..	..	1	..	6	·712	6	·712
Diseases of the respiratory organs, exclusive of consumption.	Asthma.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	·118	16	1·898
	Inflamm. of the lungs.	1	..	..	1	..	..	..	..	..	..	..	..	..	..	2	1	..	5	·593		
	Inflamm. of the chest.	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	2	·237		
	Diseased lungs.	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	·118		
	Hydrothorax.	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1	·118		
	Disease of the chest.	3	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	5	·593		
	Sore throat.	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1	·118		
Diseased liver.		1	..	..	..	..	..	..	..	..	..	..	4	..	..	..	..	..	5	·593	5	·593
Bowel complaints.	Dysentery.	..	..	..	1	..	..	..	..	1	..	..	1	..	..	..	..	..	3	·356	10	1·186
	Diarrhœa.	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	..	..	2	·237		
	Enteritis.	..	..	..	..	..	2	..	..	1	..	..	..	..	..	..	1	..	4	·474		
	Ulcerationof the bowels	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	·118		
Other diseases of the abdomen.	Obstructn.of the bowels	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	·237	4	·474
	Diseased stomach.	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1	·118		
	Peritonitis.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	·118		
Diseases of the urinary organs.	Diseased bladder.	2	..	..	..	..	1	..	..	..	..	..	1	..	..	..	..	..	4	·474	7	·830
	Inflam. of the bladder.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	·118		
	Stricture of urethra.	..	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	2	·237		
Dis. of the gen. appar.	Puerperal fever.	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	·118	2	·237
	Childbirth.	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1	·118		
Other diseases not classified.	Hæmorr. in abdomen.	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	·118	7	·830
	Dropsy.	1	..	..	..	..	..	..	2	..	..	..	1	..	..	..	..	..	4	·474		
	Gangrene of foot.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	2	·237		
Diseases of undefined nature.	Debility.	..	..	..	..	..	..	..	2	..	..	..	..	1	..	..	..	..	3	·356	11	1·305
	Decay of nature and old age.	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	·237		
	Broken constitution.	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	2	·237		
	Mass of disease.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	·118		
	Accidental injury.	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	2	·237		
	Found dead in bed.	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	·118		
Suicide.		..	..	..	1	1	3	..	1	..	..	..	5	..	..	..	1	..	12	1·423	12	1·423

TABLE XXII.—Showing the mortality caused by different diseases amongst the criminal Prisoners in the Wakefield House of Correction in the nine years, 1835 to 1843.

Years.		1835	1836	1837	1838	1839	1840	1841	1842	1843	Whole period.	Mortality per 1,000 from each disease.	Deaths from each class of diseases.	Mortality per 1,000 from each class of diseases.
Average no. of prisoners.		415	354	423	506	495	606·9	677	408·3	769	5,050			
Deaths.		12	10	14	24	12	21	17	14	11	135	26·732	135	26·732
Fevers.	Typhus fever.	3	1	1	10	...	5	2	4	2	28	5·554	29	5·742
	Debility after severe fev. & diar.	...	...	...	1	...	...	...	...	...	1	·198		
Epid. diseases not classified.	Erysipelas.	...	...	1	...	...	2	1	1	...	5	·990	6	1·188
	Influenza.	...	...	1	...	...	...	...	...	...	1	·198		
Consumption.	Consumption.	...	2	1	2	4	1	3	2	3	18	3·564	20	3·960
	Hæmoptysis.	...	1	...	...	...	1	...	...	...	2	·396		
Other tuberc. dis.	Scrofula.	...	...	...	...	...	...	...	...	1	1	·198	2	·396
	Mesenteric dis.	...	...	...	...	...	1	...	...	...	1	·198		
Diseases of the brain and nervous sys.	Effusion on, or congest. of, brain	1	...	...	...	...	3	2	...	...	6	1·188	9	1·78
	Apoplexy.	...	1	...	...	...	...	...	...	...	1	·198		
	Tetanus.	...	...	...	...	...	...	1	...	...	1	·198		
	Paralysis.	...	...	...	...	1	...	...	...	...	1	·198		
Diseases of the heart.	Diseased heart and dropsy.	1	...	...	...	...	...	...	...	...	1	·198	3	·59
	Angina pectoris.	...	...	...	...	1	...	...	...	...	1	·198		
	Hydropericard.	...	...	...	...	...	...	1	...	...	1	·198		
Diseases of the respiratory organs, exclus. of consumpt.	Chro. laryngitis.	...	...	...	1	...	...	...	...	...	1	·198	29	5·75
	Bronchitis.	...	...	3	3	...	2	2	2	...	12	2·376		
	Pneumonia.	1	1	1	...	1	...	...	...	...	4	·792		
	Inflam. of chest.	1	...	...	...	...	...	...	...	...	1	·198		
	Pleurisy.	1	...	...	...	...	2	...	...	...	3	·594		
	Hydrothorax.	...	...	...	1	1	...	1	1	2	6	1·188		
	Congest. of lungs	...	...	...	...	...	...	1	...	...	1	·198		
	Diseased lungs.	...	...	...	...	...	...	...	...	1	1	·198		
Jaundice.		...	...	...	...	...	1	...	...	...	1	·198	1	·198
Bowel complaints.	Dysentery.	...	...	1	1	1	...	1	...	...	4	·792	16	3·16
	Diarrhœa.	1	2	1	...	...	1	...	...	1	6	1·188		
	Cholera.	...	...	...	...	...	...	...	1	...	1	·198		
	Enteritis.	...	1	...	1	...	1	...	...	...	3	·594		
	Ulceration of bowels.	1	...	...	1	...	...	...	...	...	2	·396		
Other diseases of the abdomen.	Gastritis.	1	...	...	...	...	...	...	...	...	1	·198	2	·39
	Peritonitis.	...	...	...	...	...	1	...	...	...	1	·198		
Other diseases not classified.	Dropsy.	1	...	1	...	3	...	...	...	...	5	·990	10	1·98
	Atrophy and marasmus.	...	...	1	1	...	...	1	...	...	3	·594		
	Phagedœna of leg with debility.	...	...	...	...	...	...	...	1	...	1	·198		
	Diseased joint.	...	...	...	...	...	...	...	1	...	1	·198		
Diseases of undefined *nature.*	Debility.	...	1	2	1	...	...	...	...	1	5	·990	6	1·18
	Debility from diseased heart and stomach.	...	...	...	...	...	...	...	1	...	1	·198		
Violent deaths.	*Suicide.*	...	...	...	...	...	...	1	...	...	1	·198	2	·39
	Accidental suffocation.	...	...	...	1	...			...	...	1	·19		

TABLE XXVI.—Showing the mortality caused by different diseases amongst the prisoners (both whi and coloured) in the Eastern Penitentiary, Pennsylvania, during the fourteen years, 1830 to 1843.

Years.		1830	1831	1832	1833	1834	1835	1836	1837	1838	1839	1840	1841	1842	1843	Whole period.	Mortality per thousand prisoners from each disease.	Deaths from each class of diseases	Mort lity p thou sand prison ers fro each class of dis eases
Average daily number of prisoners.		31	67	91	123	183	286	360	386	402	418	394	335	331	359	3,737			
Fevers.	Cont. fev.	...	...	...	...	...	...	...	1	...	...	...	...	1	...	2	·535	4	1·07
	Remit. fev.	...	...	...	...	1	...	...	1	...	...	...	...	...	...	2	·535		
Epd. dis. not classified.	Erysipelas	...	...	...	...	...	...	...	...	...	...	...	...	...	1	1	·267	3	·80
	w. syphil.																		
	Smallpox.	...	...	...	...	...	...	...	...	2	...	...	...	...	...	2	·535		
Consumption.	Consumpt.	1	...	1	...	2	2	3	6	7	1	12	4	2	7	48	12·844	61	16·32
	Chron. infl. lgs. w. scro.	...	...	...	...	...	...	...	3	3	...	...	...	...	...	6	1·605		
	Ch. pulmo. dis. wh. scf.	...	...	...	...	...	...	...	...	...	...	...	5*	...	...	5	1·337		
	Brain fev. withscrof. &dis. lngs.	...	...	...	...	...	...	...	1	...	...	...	...	...	...	1	·267		
	Scr. of chst	...	...	...	...	...	...	...	...	1	...	...	...	...	...	1	·267		
Other tubercular or scrofulous diseases.	Scrofula.	...	...	...	...	...	1	...	1	...	...	2	1	3	...	8	2·140	20	5·35
	Chro. pleu. with scro.	...	...	...	...	...	...	...	...	...	2	...	...	...	...	2	·535		
	Disd. mesent. glds	...	1	...	...	...	...	...	...	...	...	...	...	...	...	1	·267		
	Scr. perito.	...	...	...	...	...	...	...	...	1	1	1	...	...	2	5	1·337		
	Scr. perito. & pleurit.	...	...	...	...	...	...	...	...	2	1	...	...	...	...	3	·802		
	Sc. enterit.	...	...	...	...	...	...	...	...	...	...	...	...	...	1	1	·267		
Diseases of the brain and nervous system.	Brain fev.	...	...	...	...	...	...	...	...	...	2	...	...	...	...	2	·535	9	2·40
	Apoplexy.	...	...	...	1	...	...	1	...	...	...	...	...	...	...	2	·535		
	Paralysis.	...	...	...	...	1	...	1	...	...	...	...	...	...	...	2	·535		
	Tetanus.	...	...	...	...	...	...	...	...	...	...	...	...	1	...	1	·267		
	Epilepsy.	...	1	...	...	...	...	...	...	...	...	...	...	...	...	1	·267		
	Mania.	...	...	1	...	...	...	...	...	...	...	...	...	...	...	1	·267		
Enlarged heart.		...	...	...	...	...	...	...	...	...	...	...	...	1	...	1	·267	1	·26
Diseases of the respiratory organs, exclus. of consumpt.	Drop. & ast.	...	...	...	...	...	1	...	...	...	...	...	...	...	...	1	·267	29	7·76
	Infl. of lgs.	...	...	...	...	...	...	1	...	...	...	...	...	...	...	1	·267		
	Chr. infl. & dis. of lgs.	...	...	...	...	...	2	3	3	5	...	...	5*	...	...	18	4·817		
	Chr. pleur.	...	...	...	...	...	...	...	...	...	2	3	...	1	...	6	1·605		
	Hydrothx.	...	1	...	...	1	...	...	...	...	...	...	...	...	...	2	·535		
	Bronchitis	...	...	...	...	...	...	1	...	...	...	...	...	...	...	1	·267		
Bowel complaints.	Diarrhœa.	...	...	...	...	...	...	...	...	1	...	...	...	...	...	1	·267	3	·80
	Infl. of sto. & bowels.	...	...	...	...	...	...	1	...	...	...	...	...	...	...	1	·267		
	Chron. inflammat. of bowels	...	...	...	...	...	...	...	...	1	...	...	...	...	...	1	·267		
Other dis. of the abdomen.	Infl. of sto.	...	...	...	...	...	...	...	...	...	...	1	...	...	...	1	·267	3	·80
	Chro. infl. ditto.	...	...	...	...	...	...	...	...	1	...	1	...	...	...	2	·535		
Chr. cystitis w. paral.		...	...	...	...	...	...	...	...	2	...	1	...	...	...	3	·802	3	·80
Other diseases not classified.	Rheumat.	...	...	...	...	...	1	...	...	...	...	...	...	...	...	1	·267	6	1·60
	Dropsy.	...	...	...	...	...	...	...	...	...	...	...	1	...	...	1	·267		
	Syphilis.	...	...	...	...	...	...	...	1	...	...	...	...	...	...	1	·267		
	Disd. hip.	...	...	...	...	...	...	...	...	...	1	...	...	...	...	1	·267		
	Frac. neck thigh bone	...	1	...	...	...	...	...	...	...	...	...	...	...	...	1	·267		
	Hæmorr.	...	...	1	...	...	...	...	...	...	...	...	...	...	...	1	·267		
Debility.		...	...	...	...	...	...	1	...	...	1	2	...	...	...	4	1 070	4	1·07
Suicide.		...	...	1	...	...	...	...	...	...	...	...	...	...	...	1	·267	1	·26
Total Deaths.		1	4	4	1	5	7	12	17	26	11	22	17	9	11	147	39·336	147	39·33

* *In the official report for the year* 1841, ten coloured prisoners are stated to have died from "c *nic pulmonary disease combined in some instances with scrofula and syphilis." Half these cases een classed with consumption, and half with other affections of the lungs.*

TABLE XXVII.—Showing the mortality caused by different diseases and classes of diseases amongst the white prisoners in the Eastern Penitentiary, during eight years.

Years.		1830	1833	1837	1838	1839	1841	1842	1843	Whole period.	Annual mortality per thousand prisoners from each disease.	Deaths from each class of diseases.	Annual mortality per thousand prisoners from each class of diseases.
Average number of prisoners.		21·	89·30	233·	241·	245·	201·	212·	235·	1,477			
Fever.	Fever.	..	..	..	..	..	..	1	..	1	·677	1	·667
Epidemic diseases, not classified.	Smallpox.	..	..	..	2	..	..	..	..	2	1·354	3	2·031
	Erysipelas, with syphilis.	..	..	..	..	.	..	..	1	1	·677		
Consumption.	Consumption.	1	..	5	3	..	4	1	4	18	12·186	19	12·864
	Brain fev. with scrof. & dis. lungs	..	..	1	..	..	..	..	..	1	·677		
Dis. of brain and nervous system.	Brain fever.	..	..	..	..	1	..	..	..	1	·677	2	1·354
	Apoplexy.	..	1	..	..	..	..	..	..	1	·677		
Enlarged heart.		..	..	..	..	..	..	1	..	1	·677	1	·677
Chronic pleurisy.		..	..	..	..	1	..	..	..	1	·677	1	·677
Diarrhœa.		..	..	..	1	..	..	..	..	1	·677	1	·677
Chronic cystitis.		..	..	..	1	..	..	..	..	1	·677	1	·677
Syphilis.		..	..	1	..	..	..	..	..	1	·677	1	·677
All diseases.		1	1	7	7	2	4	3	5	30	20·311	30	20·311

TABLE XXIII.—Showing the mortality caused by different diseases amongst the crimi prisoners in the Devizes House of Correction in the twelve years, 1829 to 1840.

Years.		1829	1830	1831	1832	1833	1834	1835	1836	1837	1838	1839	1840	Whole period.	Mortality per 1,000 from each disease.	Deaths per 1,000 from each class of diseases.	Mo lity 1,0 fro ea cl of ea
Average number of prisoners.		161	159	126	186	233	233	186	126	159	161	164	202	2,096			
Deaths.		4	7	2	3	3	2	0	5	4	1	2	2	32	15·267	32	15·
Typhus fever.		2	3	1	...	...	...	...	2	...	1	...	...	9	4·293	9	4·
Consumption.		...	1	...	1	...	...	...	2	2	...	1	...	7	3·335	7	3·
Diseases of the brain and nervous system.	Apoplexy.	...	2	...	1	...	...	...	1	...	...	...	...	4	1·908	7	3·
	Paralysis.	...	...	1	1	...	...	...	...	...	...	...	...	2	·954		
	Hydrocephalus.	...	...	...	...	...	...	...	...	1	...	...	...	1	·477		
Dis. of respir. organs, exclus. of consumption	Inflammation of the lungs.	...	...	...	...	...	...	...	...	...	...	...	1	1	·477	2	·
	Disd. liv. & lungs	...	...	...	...	...	...	...	...	...	...	1	...	1	·477		
Bowel complaints.	Dysentery.	...	1	...	...	...	...	...	...	1	...	...	1	3	1·431	4	1·
	Enteritis.	...	...	...	...	...	1	...	...	...	...	...	...	1	·427		
Dropsy.		2	...	...	...	...	...	...	...	...	...	...	...	2	·954	2	·
Old age.		...	...	...	...	...	1	...	...	...	...	...	...	1	·477	1	·

TABLE XXIV.—Showing the mortality caused by different diseases amongst the cri minal prisoners in the Knutsford House of Correction, in the seven years 1834 to 1840.

Years.		1834	1835	1836	1837	1838	1839	1840	Whole period.	Mortality per 1,000 from each disease.	Deaths from each class of diseases	Mortality per 1,000 from each class of diseases.
Average number of prisoners.		213	213	236	232	232	266	333	1,725			
Deaths.		6	5	3	3	3	6	8	34	19·710	34	19·710
Typhus fever.		2	...	...	1	...	...	...	3	1·739	3	1·739
Consumption.	Consumption.	1	...	1	...	3	3	2	10	5·797	11	6·376
	Hæmoptysis.	1	...	...	...	...	...	...	1	·579		
Diseases of the brain and nervous system.	Diseased brain.	...	...	...	...	...	1	...	1	·579	4	2·318
	Apoplexy.	1	1	...	1	...	...	...	3	1·739		
Dis. of the respiratory organs, exclusive of consumption.	Inflammation of the lungs.	...	...	...	...	...	...	2	2	1·159	4	2·318
	Inflammation of the chest.	...	...	1	...	...	...	...	1	·579		
	Water in the chest.	...	1	...	...	...	...	...	1	·579		
Dysentery.		...	...	...	...	...	...	3	3	1·739	3	1·739
Other diseases of the abdomen.	Diseased stomach.	1	...	1	...	...	...	...	2	1·159	4	2·318
	Spasm of stomach.	...	1	...	...	...	...	...	1	·579		
	Colic.	...	...	...	1	...	...	...	1	·579		
Diabetes.		...	1	...	...	...	...	...	1	·579	1	·579
Cancer of uterus.		...	1	...	...	...	...	...	1	·579	1	·579
Other diseases not classified.	Psoas abscess.	...	...	...	...	...	...	1	1	·579	2	1·159
	Diseased knee-joint.	...	...	...	...	...	1	...	1	·579		
Broken constitution.		...	...	...	...	...	1	...	1	·579	1	·579

TABLE XXV.—Showing the mortality caused by different diseases amongst the criminal prisoners in the Lancaster County Gaol in the 19 years, 1825 to 1843.

Years.		1825	1826	1827	1828	1829	1830	1831	1832	1833	1834	1835	1836	1837	1838	1839	1840	1841	1842	1843	Whole period	Mortality per thousand from each disease.	Deaths from each class of diseases.	Mortality per thousand from each class of diseases.
Average number of prisoners.		213	248	249	172	228	251	250	247	273	289	274	169	239	298	318	340	334	340	271	5011			
Deaths.		2	10	5	12	11	8	5	3	2	5	5	5	4	8	5	3	3	3	3	102	20·355	102	20·355
Fevers.	Typhus fever.	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	..	1	4	·798	5	·997
	Bilious fever.	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	·199		
Erysipelas.		..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	2	·398	2	·398
Consumption.		1	..	1	3	4	4	2	2	1	1	1	1	1	6	3	..	1	..	..	32	6·385	32	6·385
Scrotula.		..	..	..	..	..	..	2	..	..	..	..	..	1	..	..	..	..	1	..	4	·798	4	·798
of brain rv. sys.	Effus.,orconges.of,	..	1	..	..	..	..	..	..	1	..	2	..	..	..	..	..	..	..	..	4	·798	7	1·395
	Paralysis. [brain.	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	1	..	3	·597		
he re-	Asthma.	..	..	..	..	..	1	..	..	..	..	1	..	..	1	..	..	..	..	..	3	·597	11	2·192
y or-	Diseased lungs.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	·199		
lus. of	Hydrothorax.	1	1	..	3	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	6	1·196		
ption.	Bronchitis.	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	·199		
Diseased liver.		..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	·199	1	·199
Bowel complaints.	Dysentery.	..	4	1	5	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10	1·993	16	3·193
	Diarrhœa.	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	·398		
	Cholera.	..	1	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	3	597		
	Ulcerat. of bowels.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	·199		
Other dis. of the abdomen.	Diseased stomach	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	·199	2	·398
	Hernia.	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	·199		
Diabetes.		..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	·199	1	·199
Other diseases not classified.	Dropsy.	..	..	..	..	3	..	..	..	..	..	..	1	1	..	..	..	..	..	..	5	·997	6	1·196
	Abscess.	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	·199		
Diseases of undefined nature.	Debility.	..	..	..	..	1	..	..	1	..	..	..	1	1	..	..	1	..	1	1	7	1·395	15	2·993
	Atrophy or maras-	..	1	2	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	5	·997		
	Cachexia. [mus.	..	..	..	..	1	..	..	..	..	1	..	..	..	..	..	..	..	..	..	2	·398		
	Found dead in bed.	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	·199		

TABLE XXX.—Showing the mortality caused by different diseases and classes o diseases amongst the prisoners in the Auburn State Prison (New York) during thirteen years.

Years.		1826	1827	1828	1829	1830	1831	1832	1533	1834	1836	1837	1839	1840	Whole period.	Annual mortality per thousand prisoners from each disease	Deaths from each class of diseases.	Annual mortality per thousand prisoners from each class of diseases
Number of prisoners at the end of each year.		427	525	571	639	620	647	683	679	679	648	705	670	695	8,188			
Fevers.	Fever.	...	2	1	1	...	...	...	...	...	...	2	1	1	8	·977	9	1·099
	Remitt. fever.	...	...	...	...	...	...	...	...	...	1	...	...	...	1	·122		
Erysipelas.		...	...	...	...	...	...	...	...	...	...	...	1	1	2	·244	2	·244
Consumptn.	Consumption.	4	4	5	2	10	12	9	4	2	8	5	5	7	77	9·402	78	9·526
	Hæmoptysis.	...	1	...	...	...	...	...	...	...	...	...	...	...	1	·122		
Scrofula.		...	...	...	...	1	...	...	1	...	...	...	...	1	3	·366	3	·366
Diseases of the brain & nervous system.	Chron. inflammation of the brain.	...	...	2	...	...	...	2	...	...	...	...	...	...	4	·488	8	·977
	Hydrocephalus	...	...	...	...	1	...	...	...	...	...	...	...	...	1	·122		
	Apoplexy and paralysis.	...	...	...	...	...	...	...	...	1	1	...	...	...	2	·244		
	Mania.	...	...	...	...	1	...	...	...	...	...	...	...	...	1	·122		
Dis. of heart.	Diseased heart.	...	1	...	...	...	...	...	...	...	...	...	...	...	1	·122	2	244
	Hydrops peric.	1	...	...	...	...	...	...	...	...	...	...	...	...	1	·122		
Diseases of the respiratory organs, exclusive of consumptn.	Inflammation of lungs.	1	...	...	...	1	1	...	...	2	3	4	...	1	13	1·587	22	2·687
	Asthma, and Asthma with Dropsy.	...	...	...	...	...	...	...	...	...	3	1	...	...	4	·488		
	Hydrothorax.	...	...	...	...	...	...	...	...	1	...	1	1	...	3	·366		
	Quinsey.	...	...	...	...	...	...	...	...	1	...	...	...	...	1	·122		
	Bronchitis.	...	...	...	...	...	...	...	...	...	...	1	...	...	1	·122		
Hepatic diseases.	Chronic hepatitis.	...	...	...	...	...	1	...	...	...	...	1	...	...	2	·244	3	·366
	Chron. abscess of the liver.	...	...	...	...	...	...	...	1	...	...	...	...	...	1	·122		
Bowel complts.	Dysen. & Diarr.	...	1	...	...	...	...	...	...	1	1	...	...	...	3	·336	5	·610
	Enteritis.	...	...	...	...	1	...	...	...	1	...	...	...	...	2	·244		
Other dis. of abdomn.	Infla. of stom.	1	...	...	1	...	...	...	...	1	1	...	...	...	4	·488	6	·732
	Hœmatemesis	...	...	...	1	...	...	...	...	...	...	...	...	...	1	·122		
	Peritonitis.	...	...	...	...	1	...	...	...	...	...	...	...	...	1	·122		
Scirrhus of the testes.		...	...	...	...	...	...	...	1	...	...	...	...	...	1	·122	1	·122
Other diseases not classified.	Dropsy.	...	...	...	...	...	...	...	1	...	...	1	...	1	3	·366	8	·977
	Syphilis.	...	...	...	...	...	...	...	1	...	...	...	...	...	1	·122		
	Diseased spin.	...	...	...	...	...	...	...	1	...	...	...	...	...	1	·122		
	Lumbar abscs.	...	...	...	...	...	...	...	...	...	...	...	1	...	1	·122		
	Fever after an operation.	...	...	...	...	...	...	1	...	...	...	...	...	...	1	·122		
	Hæmorrhage.	...	...	...	...	1	...	...	...	...	...	...	...	...	1	·122		
Dis. of indefin. nature.	Atrophy and gen. debility.	...	...	...	...	1	...	...	...	...	...	2	...	...	3	·366	4	·488
	Hectic fever.	...	...	1	...	...	...	...	...	...	...	...	...	...	1	·122		
Violent deaths.	Compound fracture of the leg.	...	...	...	...	...	...	...	1	...	...	...	...	...	1	·122	8	·977
	Suicide.	...	...	...	1	...	1	...	...	...	...	1	...	2	5	·610		
	Accidental suffocation.	...	...	...	...	...	...	...	...	...	...	...	1	...	1	·122		
	Homicide.	...	...	...	...	...	...	...	...	1	...	...	...	...	1	·122		
Deaths from all causes.		7	9	9	6	18	15	12	11	11	18	19	10	14	159	19·418	159	

TABLE XXXI.—Showing the mortality caused by different diseases amongst the prisoners in the Charlestown State Prison (Massachusetts) during eleven years.

Years.*		1829	1831	1832	1833	1834	1835	1836	1837	1838	1839	1840	Whole period.	Mortality per thousand prisoners from each disease.	Deaths from each class of diseases.	Mortality per thousand prisoners from each class of diseases.
Average number of prisoners.		262	256	227	250	277	279	278	291	303	318	318	3,059			
Fever.		..	..	1	1	2	1	..	..	..	1	..	6	1·961	6	1·961
Consumption.		4	4	7	3	2	2	2	3	3	2	1	33	10·787	33	10·787
Diseases of the brain and nervous system.	Apoplexy.	..	..	..	..	..	..	1	..	..	..	..	1	·326	3	·980.
	Paraplegia.	..	1	..	..	..	..	..	..	..	..	..	1	·326		
	Mania.	..	1	..	..	..	..	..	..	..	..	..	1	·326		
Ossification of arteries.		..	..	..	..	..	..	..	..	..	1	..	1	·326	1	·326
Diseases of the respiratory organs, exclusive of consumption.	Pneumonia.	..	..	..	..	..	..	..	..	1	..	..	1	·326	5	1·634
	Hydrothorax.	..	..	..	1	..	..	..	1	..	..	1	3	·980		
	Empyema.	..	..	..	..	..	..	1	..	..	..	..	1	·980		
Chronic diarrhœa.		2	..	..	..	..	..	..	..	..	..	..	2	·653	2	·653
Other diseases not classified.	Dropsy.	..	..	2	..	..	..	..	..	..	..	..	2	·653	4	1·307
	Syphilis.	..	1	..	..	..	..	..	..	..	..	..	1	·326		
	Abscess.	..	..	..	..	..	..	..	1	..	..	..	1	·326		
Diseases of undefined nature.	Cachexia.	..	..	..	..	..	..	..	..	..	1	..	1	·326	4	1·307
	Old age.	..	..	1	..	..	..	..	..	2	..	..	3	·980		
Suicide.		..	..	..	1	..	..	..	..	1	..	..	2	·653	2	·653
Deaths from all causes.		6	7	11	6	4	3	4	5	7	5	2	60	19·614	60	19·614

* The causes of death during the year 1830 are not stated in the Reports.

TABLE XXVIII.—Showing the mortality caused by different diseases and classes of diseases amongst the coloured prisoners in the Eastern Penitentiary, during eight years.

Years.		1830	1833	1837	1838	1839	1841	1842	1843	Whole period.	Annual mortality per thousand prisoners from each disease.	Deaths from each class of diseases.	Annual mortality per thousand prisoners from each class of diseases.
Average number of prisoners.		9·19	33·70	154	151	173	134	119	124	907·89			
Fevers.	Fever.	..	..	1	..	..	..	..	..	1	1·101	2	2·202
	Remittent fever.	..	..	1	..	..	..	..	..	1	1·101		
Consumption.	Consumption.	..	..	1	4	1	..	1	3	10	11·013	22	24·227
	Chron. inflam. of lungs with scrof.	..	..	3	3	..	..	..	..	6	6·607		
	Chron. pulmonary dis. with scrof.	..	..	..	..	..	5*	..	..	5	5·506		
	Scrofula of the chest.	..	..	..	1	..	..	..	..	1	1·101		
Other tubercular or scrofulous diseases.	Scrofula.	..	..	1	..	..	1	3	..	5	5·506	15	16·521
	Chronic pleurisy, with scrofula.	..	..	..	..	2	..	..	..	2	2·202		
	Scrofulous peritonitis.	..	..	..	1	1	..	..	2	4	4·405		
	Scrof. peritonitis and pleuritis.	..	..	..	2	1	..	..	..	3	3·303		
	Scrof. enteritis.	..	..	..	..	..	..	..	1	1	1·101		
Dis. of brain and nervous system.	Brain fever.	..	..	..	..	1	..	..	..	1	1·101	2	2·202
	Tetanus.	..	..	..	..	..	..	1	..	1	1·101		
Dis. of the respir. org. excl. of cons.	Chron. inflam. & disorg. of lungs	..	..	3	5	..	5*	..	..	13	14·316	15	16·521
	Chronic pleurisy.	..	..	..	..	1	..	1	..	2	2·202		
Bowel compts.	Chron. inflam. of the bowels.	..	..	..	1	..	..	..	..	1	1·101	1	1·101
Other dis. of the abdomen.	Inflammation of stomach.	..	..	..	..	..	1	..	..	1	1·101	2	2·202
	Chronic ditto.	..	..	..	1	..	..	..	..	1	1·101		
Chronic cystitis, with palsy.		..	..	..	1	..	..	..	..	1	1·101	1	1·101
Other diseases not classified.	Dropsy.	..	..	..	..	..	1	..	..	1	1·101	2	2·202
	Diseased hip.	..	..	..	..	1	..	..	..	1	1·101		
Debility.		..	..	..	..	1	..	..	..	1	1·101	1	1·101
All diseases.		..	..	10	19	9	13	6	6	63	69·372	63	69·372

* See note to Table XXVI.

Table XXIX.—Showing the mortality caused by different Diseases amongst the prisoners in the *Sing Sing* State Prison (New York) from the 1st October 1829 to the 30th September 1840.

Years.		1830	1831	1832	1833	1834	1835	1836	1837	1838	1839	1840	Whole period.	Annual mortality per thousand prisoners from each disease.	Deaths from each class of diseases.	Annual mortality per thousand prisoners from each class of diseases
Number of prisoners on the 30th September.		810	980	832	811	843	796	726	781	824	805	827	9,053			
Fevers.	Typhus or congestive fever.	1	1	6	1	1	...	1	6	5	6	...	28	3·093	28	3·093
Asiatic cholera.		...	...	103	...	...	...	...	...	...	...	...	103	11·377	103	11·377
Epid. diseases not classified.	Smallpox.	...	...	...	1	...	3	...	...	...	...	...	4	·442	5	·552
	Erysipelas.	...	...	...	...	...	...	...	...	...	1	...	1	·110		
Consumption.	Consumption.	4	9	12	10	8	15	5	5	10	8	5	91	10·051	93	10·271
	Hæmoptysis.	...	...	1	...	...	...	...	1	...	...	...	2	·220		
Scrofula.		...	3	1	...	...	...	1	...	1	1	2	9	·994	9	·994
Diseases of the brain and nervous system.	Apoplexy.	...	1	...	...	...	...	...	...	2	...	...	3	·331	11	1·215
	Paralysis.	1	...	...	...	...	...	...	...	1	...	...	2	·220		
	Epilepsy.	...	...	...	1	1	...	...	1	2	...	...	5	·552		
	Insanity.	...	...	...	...	...	...	...	...	...	...	1	1	·110		
Diseases of the heart.	Hydrops pericar.	...	...	...	...	1	...	...	...	...	...	...	1	·110	2	·220
	Aneurism.	1	...	...	...	...	...	...	...	...	...	...	1	·110		
Diseases of the respiratory organs, exclusive of consumption.	Pneumonia typhoides.	...	...	...	...	...	...	...	...	...	...	1	1	·110	14	1·546
	Hydrothorax	3	1	...	1	1	...	2	1	1	1	...	11	1·215		
	Adhes. of lungs.	...	...	...	...	...	...	...	...	...	...	1	1	·110		
	Asthma.	...	...	...	...	...	...	...	...	...	...	1	1	·110		
Hepatic diseases.	Diseased liver.	...	2	...	1	...	...	...	...	...	...	...	3	·331	7	·773
	Jaundice.	...	1	...	...	...	...	1	...	2	...	...	4	·442		
Bowel complaints.	Cholera.	...	...	...	...	...	...	...	...	...	1	...	1	·110	44	4·860
	Diarrhœa.	1	...	4	...	3	2	...	2	1	...	1	14	1·546		
	Dysentery.	3	2	4	1	...	1	1	...	5	7	...	24	2·651		
	Enteritis.	...	...	...	1	...	1	...	1	...	2	...	5	·552		
Other diseases of the abdomen.	Gastritis.	...	...	...	...	...	...	...	...	...	...	1	1	·110	3	·331
	Organic disease of stomach.	...	...	...	1	...	...	...	...	...	...	...	1	·110		
	Colic.	...	1	...	...	...	...	...	...	...	...	...	1	·110		
Other diseases not classified.	Dropsy.	...	5	11	3	2	4	...	2	2	4	2	35	3·866	41	4·529
	Syphilis and ulcers.	...	...	1	...	...	...	...	...	...	...	...	1	·110		
	Leprosy.	...	...	...	...	...	...	...	...	...	1	...	1	·110		
	Internal abscess.	...	...	...	1	...	...	...	1	...	1	...	3	·331		
	Ulcer.	1	...	...	...	...	...	...	...	...	...	...	1	·110		
Diseases of undefined nature.	Debility.	...	...	...	...	...	...	...	...	...	...	1	1	·110	2	·220
	Diseases not named.	...	...	...	1	...	...	...	...	...	...	...	1	·110		
Violent deaths.	Suicide.	1	...	...	2	1	3	...	...	1	1	...	9	·994	16	1·766
	Accident.	...	2	2	...	...	2	...	...	...	...	1	7	·773		
Deaths from all causes.		16	28	145	25	18	31	11	20	33	34	17	378	41·754	378	41·754

TABLE XXXII.—Showing the mortality caused by different diseases in the Baltimore Penitentiary (Maryland) during five years.

Years.		1831	1832	1833	1834	1837	Aggregate of the 5 years.	Mortality per thousand prisoners from each disease.	Deaths from each class of diseases.	Mortality per thousand prisoners from each class of diseases.
Average daily number of prisoners.		370	269	356	369	391	1755			
Asiatic cholera.		..	17	..	..	..	17	9·686	17	9·686
Consumption.	Consumption.	19	2	14	8	6	49	27·920	50	28·490
	Hæmoptysis.	..	1	..	..	..	1	·569		
Diseases of the brain and nervous system.	Inflamm. of brain.	..	..	1	..	..	1	·569	1	·569
Hydrops pericardii.		..	1	..	..	..	1	·569	1	·569
Lymphathic bilious catarrh.		..	1	..	..	..	1	·569	1	·569
Hepatitis.		..	..	1	..	..	1	·569	1	·569
Bowel complaints.	Chronic diarrhœa.	..	..	..	..	1	1	·569	3	1·709
	Dysentery.	2	..	..	..	..	2	1·139		
Other diseases of the abdomen.	Gastritis.	..	..	..	1	..	1	·569	3	1·709
	Hernia.	..	..	..	1	..	1	·569		
	Abdominal dis.	..	..	..	..	1	1	·569		
Other diseases not classified.	Dropsy.	1	..	..	..	..	1	·569	2	1·139
	Rheumatism.	1	..	..	..	..	1	·569		
Diseases of undefined nature.	Acute disease.	..	..	..	..	4	4	2 279	6	3·418
	Drinking cold water, when heated, and diseased lungs.	..	..	..	..	1	1	·569		
	Debility.	1	..	..	..	..	1	·569		
All causes.		24	22	16	10	13	85	48·433	85	48·433

TABLE XXXIII.—Showing the number of deaths, and annual rate of mortality caused by different diseases and different classes of diseases in the *Geneva* Penitentiary from the 1st of January 1826 to the 31st December 1841.

Average number of prisoners.... 57 Aggregate of the annual average numbers during sixteen years ..... 909·78		Number of deaths from each disease.	Annual mortality per thousand prisoners from each disease.	Number of deaths from each class of diseases.	Annual mortality per thousand prisoners from each class of diseases.
Fever (continued).		1	1·099	1	1·099
Consumption.		9	9·892	9	9·892
Other tubercular diseases.	Tubercular peritonitis.	1	1·099	2	2·198
	Scrofula.	1	1·099		
Diseases of the brain and nervous system.	Apoplexy.	1	1·099	5	5·496
	Paralysis.	2	2·198		
	Insanity.	2	2·198		
Dis. of the respir. organs, excl. of consumption.	Catarrh and sudden death in an old man.	1	1 099	4	4·396
	Chronic bronchitis.	3	3·297		
Chronic diarrhœa.		1	1·099	1	1·099
Contraction of the stomach.		1	1·099	1	1·099
Suffocation from goitre.		1	1·099	1	1·099
All causes.		24	26·380	24	26·380

TABLE XXXIV.—Showing the mortality caused by different diseases in the Penitentiary of *Lausanne*, during the ten years and eight months from May 1st, 1826, to January 1st, 1837.

Average number of prisoners.... 83 Aggregate of the annual averages for whole period 882		Number of deaths from disease.	Annual mortality per thousand prisoners from each disease.	Number of deaths from each class of diseases.	Annual mortality per thousand prisoners from each class of diseases.
Consumption.		6	6·802	6	6·802
Disaeses of the brain and nervous system.	Apoplexy.	2	2·267	5	5·669
	Congestion of brain.	2	2·267		
	Arachnitis.	1	1·134		
Diseased heart.		2	2·267	2	2·267
Diseases of the resp. organs, exclus. of consumption.	Hydrothorax.	1	1·133	2	2·267
	Affection of chest.	1	1·133		
Bowel complaints.	Dysentery.	1	1·133	2	2·267
	Chronic enteritis.	1	1·133		
Affection of the bladder.		1	1·133	1	1·134
Other diseases not classified.	Dropsy.	2	2·267	5	5·669
	Syphilis.	2	2·267		
	Caries of backbone & ribs	1	1·133		
Diseases of undefined nature.	Old age.	1	1·133	9	10·204
	Marasmus.	8	9·070		
All causes.		32	36·281	32	36·281

TABLE XXXV.—Showing the mortality caused in 1842 by different classes of diseases amongst white persons of the ages of 10 to 70, and amongst the inhabitants belonging to the African and other dark races above the age of 10 years, in the city and county of New York. The mortality per 1,000 is calculated from the census of 1840, for the dark inhabitants, and from Dr. Griscom's* estimate of the population in 1842, for the whites.

	Absolute no. of deaths in 1842.				Mortality per thousand living.					
Races.	Whites.		Blacks.		Whites.			Blacks.		
Ages.	10 to 70.		Above 10 years of age.		Males.	Females.	Both sexes.	Males.	Females.	Both sexes.
Sexes.	Males.	Fem.	Males.	Fem.						
Fevers.	149	154	10	19	1·380	1·299	1·338	1·866	2·462	2·294
Epidemic diseases, not classified.	84	57	11	8	·778	·481	·622	2·053	1·037	1·453
Consump. & Hæmop.	508	537	58	58	4·706	4·531	4·107	10·825	7·515	8·871
Other tuberc. diseases.	18	11	1	5	·167	·093	·128	·186	·647	·458
Diseases of brain and nervous system.	234	179	11	22	2·167	1·510	1·823	2·053	2·850	2·523
Diseases of the heart.	51	48	5	8	·472	·405	·437	·933	1·037	·994
Dis. of the resp. org. excl. of con.& hæmop.	150	150	26	22	1·389	1·265	1·324	4·852	2·850	3·666
Hepatic diseases.	34	38	2	4	·315	·320	·317	·373	·515	·458
Bowel complaints.	101	133	3	10	·935	1·112	1·033	·559	1·295	·994
Other dis. of the abd.	26	60	3	1	·241	·506	·335	·559	·129	·305
Dis. of the urinary organs.	13	6	..	..	·120	·050	·083			
Dis. of the generative organs.	1	90	..	5	·009	·759	·401	..	·647	·382
Other diseases, not classified.	84	94	5	12	·778	·793	·781	·933	1·555	1·300
Diseases of undefined nature.	25	17	7	13	·231	1 43	·185	1·306	1·684	1·529
Violent deaths.	139	51	10	11	1·288	·430	·834	1·866	1·425	1·606
All causes.	1617	1625	152	198	14·980	13·712	14·317	28·369	25·654	26·776
Causes not specified.	8	14	4	2	·074	·118	·097	·746	·258	·458
Total deaths.	1625	1639	156	200	15·054	13·814	14·405	29·115	25·913	27·225

* *See the Annual Report* of the Interments in the City and County of *New York, for 1842,* by John H. Griscom, M.D.

TABLE XXXVI.—Showing the mortality per thousand living, amongst the dark inhabitants of New York, above the age of ten years; calculated from other data than those on which the estimate in Table XXXV. is founded.

		Mortality per thousand from consumption and hæmoptysis.	Mortality per thousand from marasmus and scrofula.	Mortality per thousand from all diseases.
Calculated from the population of 1840, and the deaths in 1838.	Males.	9·891	·933	31·732
	Females.	7·644	·388	22·933
Calculated from the population of 1840 and the mean of the deaths in 1838 and 1842.	Males.	10·358	·559	30·421
	Females.	7·579	·518	24·423

TABLE XXXVII.—Number of the white inhabitants of New York from 10 to 70 years of age, and of the dark inhabitants above the age of 10 years.

	White Males.	White Females.	Black Males.	Black Females.
In 1840 according to the census of the United States.	103,885	112,913	5,358	7,718
In 1842 according to Dr. Griscom's computation.	107,941	118,502		

OBSERVATIONS

ON CLEFT PALATE,

AND ON

STAPHYLORAPHY.

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The subjects to which the following observations refer, have attracted considerable attention among surgeons during the last five-and-twenty years, since Graefe and Roux in Europe, and Warren in America, showed that the congenital deformity might be successfully treated on principles similar to those which guide the practitioner in the management of hare-lip. It seems to me doubtful, however, whether our own countrymen have displayed equal zeal with our continental and transatlantic brethren in this department of surgery. It is true that most of our modern surgical authorities have taught the operation of staphyloraphy, and also that it has been performed frequently in different parts of Britain. Yet since it was first done in England by Mr. Alcock, in 1821, it cannot be said that the amount of either individual or collective experience on this proceeding is in just proportion to that acquired by surgeons in other parts of the world. It is known that more

than two years ago, the distinguished author of the operation, Roux, had performed it upwards of one hundred times, with a most satisfactory amount of success,—two thirds of the simple cases, and one third of those which were complicated, having derived benefit from the proceeding. In 1843, Dr. Mütter, of Philadelphia, had operated twenty-one times upon the soft and hard palate, and out of this number "had failed to relieve the patient but in two cases;" and up to the same year, Dr. J. Mason Warren, of Boston, had treated fourteen cases, in thirteen of which he had obtained most signal advantages from his judicious attempts to close the fissure by the operation in question. I am not aware that equal success has been achieved by any surgeon in these islands; and as far as my own personal experience extends, the results have been, up to a recentdate, so unsatisfactory that I have had little confidence in recommending the operation.

The condition of congenital defective palate, and the various operations which have been proposed to remedy such an evil, have not, so far as I am aware, been brought under the notice of this Society. The names of Graefe, Roux, Warren, Dieffenbach, Brodie, Guthrie, Liston, Bushe, Cusack, and Crampton, guarantee that the subjects have been sufficiently interesting and important to attract some of the leading talent of the day, and I am not without hope that the purport of this paper will serve as my apology for occupying a portion of the time of this meeting.

The fissure in cleft palate may be such as only to divide the uvula, or it may extend forwards through the soft and hard parts as far as the lips, in which latter instance there is generally a hare-lip as well. In the uvula, soft palate, and even through the palate bones, as also a portion of the superior maxillæ, the fissure is invariably in the mesial line, but when the alveoli in front participate in the malformation, it is somewhat to one side. In certain instances the fissure is double in front, when the whole of it may be likened to the letter Y; the two lines in front leaving between them the intermaxillary bone.

When there is merely a bifid uvula, there are not any of those distressing circumstances which accompany the more extended fissure, and in such instances, no demand is made on the skill of the surgeon, or that of the artificer in obturators; but when the gap is longer, it is well known how eagerly the unfortunate party will have recourse to an apparatus, or submit to any operation, which may promise relief, as regards the tone of the voice and deglutition.

On the present occasion I shall only advert to the advantages which may be obtained in instances of single or double fissure extending through the entire hard palate, and when accompanied with hare-lip, by the judicious application of compresses, and also by the early performance of the operation for hare-lip. It is well-known that a gap, however wide, may, if treated in early life, be brought to the con-

dition of a narrow chink, by the means alluded to; but we have no control over the fissure in the soft parts, save an artificial palate, unless by the operation of staphyloraphy.

Until within a very recent date, the cleft in the hard palate has been deemed beyond the reach of surgical skill, except by means of obturators, or by plastic operations, secondary to successful attempts upon the soft parts. Dr. J. M. Warren has shown, however, that the cleft, even in the hard palate, may be closed by an operation somewhat similar to that performed on the soft velum, but the proceeding has not, so far as I know, attracted any more than casual notice in this country. The remarkable feature in this gentleman's operation is, that he dissects the soft tissues from the bones on each side of the fissure, carrying his knife towards the alveoli to such an extent as to make a flap sufficiently broad to join its fellow in the mesial line; he then pares the edges of the cleft in the soft palate, and stitches the whole wound between the uvula and the anterior extremity, wherever it may be. If union takes place, the entire fissure is closed, and Dr. Warren has not alluded to any inconveniences or evil results from thus denuding the bones. Doubtless a considerable amount of reunion takes place, and towards the inner margins of the bones, as also on the upper surface of the soft portion in the middle, there will be a cicatrix, analogous to mucous membrane. Taking the vault of the hard palate in its natural condition, as being nearly semi-

circular, it is evident that by bringing down the soft parts on each side towards the tongue, ample flaps may be obtained, so that there would be little or no dragging by the stitches. In the cleft condition, this proposition is still more apparent, especially if the malformation extends through the alveoli, for then the surfaces of the hard palate slope upwards like the two sides of a triangle, and nearly join in the floor of the nostril.

Few have had the opportunity of dissecting a cleft palate, and some notice of a specimen in my possession will form an appropriate introduction to the views developed in this paper. The fissure in this instance implicates a portion of the hard as well as the whole soft palate, and is such as the surgeon frequently meets with in practice. The specimen was procured in the dissecting-room from the mouth of an aged female subject.

In the examination of this preparation there are several marked differences between it and the parts in a more natural state. The superior constrictor muscle is more fully developed than under ordinary circumstances, and its upper margin, extending between the basilar process of the occipital bone and the internal pterygoid plate, is particularly distinct. This part of the muscle forms a sort of semicircular loop, in which the levator palati muscle seems to be suspended.

The pharynx has been laid open by a perpendicular incision through the constrictors in the mesial line, and the moveable portion of the palate has been

dissected on one side. The circumflexus, or tensor palati, differs little from the natural condition, and the levator palati is much as it is usually met with, its lower end spreading out in all directions on the soft palate. The palato-pharyngeus consists of two distinct bundles of fibres; one, the smaller of the two, running between the tensor and levator palati; the other, a mass equal in size to a goose quill, seems to form the principal part of the free portion of the palate; and posteriorly its fibres, previous to joining those of the other bundle, form the whole muscular portion of the posterior pillar of the fauces. This muscle arises by tendinous and fleshy fibres from the posterior margin of the osseous palate and the inner surface of the internal pterygoid plate, and takes its usual course and attachment posteriorly. A bundle of fibres, about the size of a crow-quill, can be traced along the lower border of the inner margin of the soft flap. These fibres extend between the posterior margin of the hard palate and the uvula, and are probably analogous to the azygos uvulæ. The palato-glossus can scarcely be distinguished. A small arterial twig, doubtless a branch of the ascending pharyngeal artery, can be traced between the levator and tensor palati muscles. The throat and upper part of the pharynx generally is smaller than in the well formed state, but the deficiency in the mesial line of the palate seems more the result of a want of union, than of the usual materials of the velum.

The act of deglutition in the natural state of the

parts, while food is passing through the upper end of the pharynx, has been a subject of considerable speculation among physiologists, especially with reference to the manner in which the communication betwixt that bag and the posterior nares is closed for the time being.

It has been pointed out by Dzondi and Müller that the palato-pharyngei muscles, when fixed in the soft tissues at their upper ends—as in the natural state of the velum—must, during contraction, tend towards the mesial line, and so by their approximation diminish the capacity of the throat. But in the cleft state there is no central fixed line, and each muscle, acting between its extreme attachments, viz. the palatine bones above and the thyroid cartilage below, must, during contraction, tend to widen the throat rather than close it. In the condition alluded to, these muscles, joined with the levatores palati, have the effect of enlarging the gap in the mesial line. It is evident that the doctrine of the above-named physiologists will not account for the closing of the aperture under these circumstances, and how then is the occlusion effected? I am not aware that it has ever been accounted for. Malgaigne,* in describing the simple fissure of the palate, has alluded to the approximation of the edges during deglutition, "by a muscular action," as he says, "of which it is difficult to give an explanation." I think that any one who looks at the preparation in my pos-

* Manuel de Médecine opératoire. Paris, 1834; p. 486.

session, can have no doubt as to this movement. The superior constrictor has evidently the power of throwing the two lateral portions of the palate forwards and inwards, so that they are forced into contact in the mesial line, and thus the back of the fissure is closed, while the constrictor is acting on the upper part of the pharynx, like a broad semicircular band. The upper border of this muscle, as it is seen in the preparation alluded to, must evidently have the effect described, and the lower fibres will act still more effectually, in consequence of there being no connection mesially to prevent them starting forwards during contraction, so as to stretch across, almost in a direct line, extending between the lateral attachments of each muscle. Some of the fibres of the middle constrictor may also aid in this movement. The palato-pharyngei muscles are thus forced into contact, and their ends, behind and below the parts so held in apposition, may then act in the manner described by Müller, while possibly the thickness of the two portions of the soft palate may be increased by the contraction of each palato-pharyngeal muscle at the points of contact. The azygos uvulæ may probably contribute to the latter effect.

It is not so much my object on the present occasion to dwell upon the physiology of deglutition, as to draw attention to the condition of cleft palate, and the operation of staphyloraphy. I shall, therefore, now proceed to notice what has hitherto been done by the surgeon with reference to these subjects,

and to state the deductions which I have myself made from a consideration of the anatomy and physiology of the parts in question.

The operation originally performed by Roux, which was the first where the attempt by such a process succeeded, consisted in paring the edges of the fissure, and bringing them together by means of stitches, so that the cut surfaces might be united by the first intention. To keep the parts as still as possible, the patient was not allowed food of any sort, for two or three days, and was cautioned against any effort to swallow—even the saliva he was charged to let flow out of his mouth. With various modifications, as to the manner of paring the edges, introducing the stitches, tying the knots, the materials for ligatures, &c., the same proceeding has been followed by all other operators in similar cases, with the addition that extra incisions have been made by some, with the intention of facilitating the approximation of the edges of the fissure. Dieffenbach* was, I believe, the first to propose a longitudinal division of the flaps, between the alveoli and stitches, a proceeding which has also been recommended by Professor Pancoast of Philadelphia. As the last-named gentleman has written the fullest account of this process with which I am acquainted, I shall give the description in his own words, premising that the principal difference between this method, and that of Dieffenbach, is, that the latter gentleman secures

* Lancet, 1834-35, vol. i. page 695.

the ligatures before he makes the additional wound. After paring the edges of the fissure, and introducing the ligatures, "when the knots were prepared for tying, but before they were finally secured, Wenzel's cataract knife was passed from before backwards, through the attached sides of the palate, to enable the two halves of the velum to come together in the middle line, as well as to divide the insertion of the palate muscles, so as to prevent them straining the sutured edges of the palate asunder."* Mr. Liston† advises that, "before the ligatures are finally secured, the parts being put on the stretch, an incision should be made, on each side towards the alveolar ridge, through the anterior surface of the velum, by which method the edges come together more easily, and the strain is taken off the threads, so that there is less risk of these making their way out by ulceration." Dr. Mettauer, of Virginia,‡ recommends methods somewhat similar to both the plans above described. He proposes to increase the breadth of the two flaps, by making a series of lunated incisions through the flaps, each about half an inch in length, along the margins of the fissure, which he causes to heal by granulation, and thereafter proceeds with the ordinary operation; or by another method, he relaxes the parts at the time of the operation, by making a longer incision on the lower surface of the palate on each side, but a little

* American Journal of Medical Science, vol. xxxii. page 71.

† Operative Surgery. Second edition.

‡ American Journal of Medical Science, vol. xxi. page 309.

nearer the mesial line than that proposed by Mr. Liston.

Roux himself has added some additional incisions to his original proceeding. When the gap is wide, especially in cases where the hard palate is defective, by transverse incisions he separates the soft palate on each side from the posterior margin of the palatine plates of the palate bones, which permits the more ready approximation of the moveable parts towards the mesial line. A similar proceeding was also recommended by Mr. Bushe.* I have already in an early part of this paper referred to the method followed by Dr. J. M. Warren† in instances of fissure of the hard palate; but he, too, deals with the soft parts behind in an unusual manner, which I must not omit to notice here. After describing his method of dissecting the soft tissues from the bones forming the roof of the mouth, he states that "it will generally be found that by seizing the soft palate with a forceps, it can be easily brought to the mesial line. If the fissure is wide and this cannot be effected, I have found the following course to be invariably followed by success:—The soft parts being forcibly stretched, a pair of long powerful French scissors, curved on the flat side, are carried behind the anterior pillars of the palates; its attachment to the tonsil and to the posterior pillar are now

* An Essay on the Operation for Cleft Palate, by G. Bushe. New York, 1836.

† New England Quarterly Journal of Medicine and Surgery. April 1843.

to be carefully cut away, on which the anterior soft parts will at once be found to expand, and an ample flap be provided for all desirable purposes."

Before adverting to the propriety of these incisions, I must request attention to certain conspicuous movements in the soft flaps, which may be noticed during the examination of the throat of a person whose palate is cleft. It matters not whether the cleft is in the soft parts only, or runs through the bones, too,—the soft tissues alone being susceptible of motion. As we look into the open mouth, the flaps may be seen under four different conditions. First. If the parts be not irritated in any way, the gap will be quite conspicuous, the lateral flaps will be distinct, and the posterior nares, with the upper end of the pharynx, will be observed above and behind. Second. If the flaps be touched, they will in all probability be jerked upwards by a motion seemingly commencing at the middle of each. Third. If the parts be further irritated, as by pushing the finger against them into the fissure, each flap is forcibly drawn upwards and outwards, and can scarcely be distinguished from the rest of the parts, forming the sides of the nostrils and throat. And, fourth. If the parts further back be irritated, as in the second act of deglutition, the margins of the fissure are forced together, by the action of the superior constrictor muscle, already described in my observations on this process, in an earlier part of the paper.

All these conditions and movements are, in my opinion, very readily accounted for. In the first instance, the parts may be deemed in a quiescent state; in the second, the levatores palati are called into play, and move the flaps as described; and in the third, these muscles act still more forcibly, and the palato-pharyngei will join in drawing the parts outwards. The fourth condition I need not again describe.

If the free margin on one side of the fissure be seized with the forceps, drawn towards the mesial line, and the flap be then irritated, it will be drawn upwards and outwards with remarkable force; this movement, it is evident, can only be effected by two muscles, the levator palati and palato-pharyngeus. These muscles, then, I consider the chief mechanical obstacles to the junction of the margins in the mesial line. Hitherto I have taken no notice of the action of the circumflexus, or tensor palati. I am inclined to think that its action is very limited, and probably, as the dissection in my possession would indicate, is greater upon the parts outside the posterior pillar, than on those contiguous to the fissure. Neither have I alluded specially to the action of the palato-glossus, because, though it might with a feeble power incline the soft palate downwards, its influence, as regards the practical view I am now taking, is completely counteracted by the more powerful muscles connected with the palate above.

We may now look to the probable immediate

effects of the incisions which have been recommended to facilitate approximation. In Mettauer's, the small wounds are made in the course of the main portion of the palato-pharyngeus muscle. It may be doubted if this muscle be much impaired in action by such a process; but even granting this, and also that the breadth of each flap is considerably increased, the levator palati is still left to drag the parts upwards and outwards. The long incision through the mucous membrane, recommended by the same party, will obviously not reach any muscular fibres of importance. The incisions by Dieffenbach and Pancoast will scarcely touch the fibres of the palato-pharyngeus, the greater portion of that muscle passing between the incisions and the median line; and here, too, the levator palati is left almost untouched, for only a few of its fibres will be reached by such a wound. The incision recommended by Mr. Liston has evidently no reference to division of muscular fibres, and both the levator and palato-pharyngeus are left untouched. In Roux's proceeding, the anterior attachment of the palato-pharyngeus is certainly cut away, but it is questionable if the act does not give easier play to the levator palati; and at best, if it does facilitate union in the mesial line, it is at the expense of a transverse gap between the bones and soft parts, which I imagine, notwithstanding Mr. Bushe's favourable report, probably continues partially open ever after. Dr. J. M. Warren, so far as I can understand his description, only sepa-

rates the anterior pillar from the tonsil and posterior pillar, and makes no allusion to the division of muscular fibres. Possibly, however, by the clipping process which he describes, some of the fibres of the palato-pharyngeus may be divided.

Dr. Pancoast, in the language already quoted, certainly proposes "to divide the insertion of the palate muscles," but, as my demonstration proves, he cannot touch the insertion of one muscle, and can only reach the other, or a part of it, by hazard; and so indefinite has been the language used by all parties, with reference to the object of these different incisions, that I believe myself justified in stating that a distinct proposal, founded on anatomical and physiological data, has never yet been made. It has been one of my objects in drawing up this paper to do so, and I have ventured to bring the whole subject under the notice of this Society, in the hope that the novelties which it contains, with reference to the anatomy and physiology, as well as the operative proceedings immediately to be considered, may attract a share of attention from English surgeons, equivalent to that exhibited in other parts of the world, and sufficient to test the value of my views and proposals in point of practical importance.

I imagine that few who have listened to this paper throughout, will have any difficulty in anticipating what my proposals will be. I therefore, without further preamble, propose, as an important accessory to the operation of staphyloraphy, that

the surgeon should, on strictly scientific grounds, and in accordance with the modern principles of myotomy, so conduct his incisions as to destroy all motory power in the soft palate for the time being, and thus permit that repose of the stretched velum which is so essential to a happy result; in other words, I advise the division of the levator palati, the palato-pharyngeus, and the palato-glossus muscles. The first of these steps I deem of the greatest importance, the second scarcely less so, and the third may be effected or not, as the circumstances seem to demand.

It will be observed that, by dividing the above-named muscles, all motory influence in an outward, upward, or downward direction, is cut off, and the only muscles which can act in anything like a direct manner upon the soft palate are the superior constrictors of the pharynx. These, however, will only act during deglutition, and even then, their tendency will be to throw the parts closer instead of separating them.

We all know that union by the first intention occasionally fails, even under circumstances the most favourable, and though not sanguine enough to suppose that what I now advance will, in all instances, insure success, I feel satisfied that my proposals answer an important desideratum. It has been remarked, during the operation of staphyloraphy, that after the edges of the fissure have been pared, there has often been difficulty in distinguishing the flaps, so completely have they been drawn to each side.

Even after this has been done, the gap, according to the proceeding which I follow, is actually less than before the operation; and in closing the wound with the stitches, there is no dragging effort required to bring the edges into apposition.

The incisions necessary for the division of the three muscles are probably less in extent than those which have been practised, and their immediate effect will also be more decided; ultimately, after the wounds have united, the re-union of fibres is so complete that any inconvenience which might be anticipated from the temporary division of the muscles is scarcely to be perceived.

Whilst writing these observations I have examined a palate which had been successfully treated, ten months ago, on the method here propounded, and the motion upwards, such as is seen in the normal palate from the action of the levator palati, as also the effects produced by the palato-pharyngeus, as well as palato-glossus, are such as may usually be seen in the parts naturally well formed.

Within the last twelve months I have operated on two cases of congenital cleft palate, in accordance with the views developed in this paper, and the results have been most satisfactory, as regards the union of the lateral portions.

The steps which I follow are these:—With a knife whose blade is somewhat like the point of a lancet, the cutting edge being about a quarter of an inch in extent, and the flat surface being bent semicircularly, I make an incision about half an inch

long, on each side of the posterior nares, a little above and parallel with the palatine flaps, and across a line straight downwards from the lower opening of the Eustachian tube,* by which I divide the levator

palati muscle on both sides, just above its attachment to the palate. Next I pare the edges of the

* The cut represents the posterior and upper surface of one edge of the soft palate. *a*, the levator palati; the dark line shows where it should be cut across. *b*, the inner bundle of fibres of the palato-pharyngeus forming the posterior pillar of the fauces; the black line indicates the place for division. *c*, the palato-glossus, with the mark for incision, if one should be deemed necessary. The tonsil lies between these two muscles. *d*, the tensor palati, the cartilaginous extremity of the Eustachian tube is in front of this letter. *e*, the posterior extremity of the inferior turbinated bone. *f*, the septum. *gg*, the uvula on each side stretched apart.

fissure with a straight blunt-pointed bistoury, removing little more than the mucous membrane; then, with a pair of long blunt-pointed curved scissors, I divide the posterior pillar of the fauces, immediately behind the tonsil, and, if it seems necessary, cut across the anterior pillar, too; the wound in each part being about a quarter of an inch in extent. Lastly, the stitches are introduced by means of a curved needle, set in a handle; and, the threads being tied so as to keep the cut edges of the fissure accurately in contact, the operation is completed. These different incisions may be made in the order here detailed, or possibly it may be found most convenient to divide the palato-pharyngeus first, next the levator palati, and then to pare the edges when the muscular action has been taken off.

Each of these steps requires some little separate notice. The first incision, it will be remarked, differs from all others hitherto proposed, and is founded on consideration of the anatomy of the parts. The levator palati, I have no doubt, is the main obstacle to the approximation of the margins, and is the principal cause of unsteadiness in the velum during the operation and after it has been accomplished. Its division may be effected through the method above recommended, but should the flap appear tense after the knife has been used, the incision may be further extended in case the muscle may not have been completely cut across. The extension of the incision, even without reference to the division of muscular fibres, will aid greatly in relaxing the sides of the

palate. In many instances, I believe that the levator muscle might be divided by a submucous incision, by plunging the blade through the mucous covering, and then moving it freely across the muscle.

The instructions already given, will, I imagine, enable the operator to reach the muscle with facility. I may add however that the incision should be made about midway between the hard palate and the posterior margin of the soft flap, just above the thickest and most prominent part of the margin of the cleft. The instrument used for this step is of peculiar construction. No ordinary surgical knife could have readily effected the purpose, as it is scarcely possible to apply the point of a common scalpel or bistoury to the part in question, excepting through the nostril in front, a proceeding which, in my opinion, would not answer so well as that recommended. The idea of this shape of knife was first suggested to me by a perusal of Dr. J. M. Warren's description of his mode of dealing with the hard palate. I use a variety of blades with long and short curves, each to suit the particular condition of the parts, and believe that these will be found the most convenient instruments for separating the soft tissues from the hard vault, according to Warren's process: indeed, I know of no others used among surgeons in this country, which could effect this purpose. In paring the edges I am indifferent as to which side I begin with. If it be the right side I usually stand behind the patient, —a favourite attitude with the dentist, and commence to cut either at the end nearest me, or that furthest

off, as may seem most convenient. Still standing behind, with the patient's head on my chest, I pare the left side from before, backwards, or the reverse, as may appear best; or, possibly, I may conduct these steps while standing in front of the patient, whose head is supported by an assistant; but I believe that it will be found very advantageous to conduct many of the steps of the operation while standing behind and looking over the face, as it were, into the mouth. In any way, if the incisions on the edges are begun at the uvular extremity, something must be done to keep the parts steady and tense, else great difficulty will be experienced in carrying the knife along. I prefer a long narrow forceps, with the blades having hook-like extremities, and either pull the points towards me or push them back, as may seem most advisable. I give preference to a straight probe-pointed bistoury, as the best instrument for this part of the operation. When the edges are pared, I am in the habit of again using the curved knife which has just been described. The edge at some parts will appear so thin that one may doubt whether union is likely to take place when two such surfaces are brought into apposition. To increase the breadth or depth of such parts, I run the point of the curved blade along the middle of the cut surface, and thus, when the edges are brought into contact, they are expanded, and the depth from the nasal surface to the lingual is increased, whereby there is greater probability of union. I have succeeded in this way in closing a portion of a gap where

the edges were originally not thicker than a sixpence. In dividing the pillars of the fauces, the uvula, or the posterior margin of the velum, should be seized with the forceps, and so drawn forwards as to put these parts on the stretch. The relaxation of the flaps will be the criterion as to the muscles being divided. Judging from the anatomy, and what I have seen in the living body, I should say that it will seldom be necessary to meddle with the anterior pillars.

After having used or examined most of the contrivances for passing the threads, I give decided preference to the curved needle set in a handle, the eye being near the point, so that the thread may be seen and laid hold of readily as soon as the parts have been transfixed. The first three or four stitches I now always introduce thus:—I pass the curved needle from below, straight upwards, being indifferent as to which side I begin with. And then, having carried the eye towards the mesial line, so that the ligature can be seized with the forceps and drawn down into the mouth, the needle is withdrawn, armed again with a more slender thread, and the same steps are repeated on the opposite side of the cleft. The thread last introduced is next fastened to the first, and then drawn back throughout its course, whereby the first thread, which is the one intended to close the gap, is brought into its proper position. After three or four stitches have been tied, should more be required, the curved needle can readily be pushed from one side to the other. I imagine that the

difficult process of introducing the threads, is greatly facilitated by the preliminary division of muscles which I recommend. Having, on some occasions, when the ligatures had been introduced before the edges were pared, (the course followed by Roux,) experienced annoyance from the threads being cut during the subsequent steps, I advise that they be not applied until the margins have been cut. A stout silk or flaxen thread is, in my opinion, preferable to the ligatures of leaden wire used by Dieffenbach, although these have been much applauded by some; and the surgeons' knot, as advised by Professor Smith of Maryland, and Dr. J. M. Warren, I have found sufficient to keep the wound close, until the second noose had been cast. The difficulty of keeping the first noose steady, has often been alluded to: the lead ligatures, by being twisted together, obviate this difficulty; and, recently, Sir Philip Crampton has proposed a most ingenious device for closing the stitches, which consists in running a small perforated bead of soft metal up along the two ends of the thread, which, when the gap is closed, are then fixed by squeezing the metal. I think, however, that the surgeons' knot answers all the purpose desired; and, in my own operation, there is so little dragging, in consequence of the flaccid condition of the palate, that I have occasionally found the common knot answer perfectly. For the same reason, too, I have not thought it necessary to resort to the use of any of those pieces of mechanism intended to facilitate the tying of a knot

in a deep-seated part; for, after the muscles of the palate have been divided, the soft flaps can be drawn downwards and forwards, with all requisite facility. I believe it better, rather to exceed the proper number of stitches than to have too few, and I especially recommend that a stitch be used close to the lower end of the uvula, as otherwise there is here a great tendency to separation.

The after-treatment which I have pursued, has been much the same as that recommended by Roux and most others. The patient has been desired not to speak or swallow for the first two or three days; the stitches have been removed, some on the second day, others not till the third, fourth, or sixth, as seemed most advisable; and generally the treatment has been conducted on ordinary principles. I have observed benefit from the use of enemata of gruel and strong soups.

With the exception of two cases recently treated by Sir Philip Crampton,* I believe that it has been the invariable custom to withhold all food for some days. That gentleman, however, permitted his patients to partake "of boiled bread and milk, custard, soups and jelly, twice or thrice a-day," and moreover the patients were not confined to bed. The latter part of the treatment has been acted on before; and, in my estimation, the generous method of the distinguished Surgeon-General is worthy of consideration, especially as it has been followed by success, and

* Dublin Journal of Medical Science, July 1st, 1843, vol. xxii.

also that the starving system, besides being occasionally followed by evil results, is that part of the treatment most distressing to the generality of patients.

It will be observed, that the large vessels and nerves passing through the base of the cranium in this vicinity are all behind and above the incisions which I recommend. In meddling with the pillars of the fauces there cannot be the smallest danger, and if the knife be properly placed above the palate there is nothing between it and the pterygoid plates of bone, but the levator and the tensor palati. I have traced, after a good injection, the ascending pharyngeal artery passing forwards between these two muscles,—doubtless the same branch as that which has been already referred to in the dissection of the cleft palate,—but have not experienced any annoyance from bleeding from this quarter, in the living body. It is well known that incisions in the mucous membrane will bleed more freely in some persons than in others, but all authorities agree that vexatious hæmorrhage has never yet happened in any case of staphyloraphy, nor should I apprehend any from the proceedings above described.

The variety of wounds may seem objectionable, but I doubt if they equal in extent such incisions as have been advised by others. These, in whatever manner they are made, must doubtless increase the inflammation that ensues. In the first example where I put my own method into execution, a small slough formed on the posterior margin of one of the flaps, and the gap opened again throughout more

than one half its extent. The result here might possibly have been occasioned in the manner alluded to, but it is more probable that it was in consequence of the patient being in an indifferent state of health at the time. Three months afterwards, the part which had opened was again operated on. The edges of the fissure were alone meddled with, for notwithstanding the double paring, they could be approximated with the utmost facility. The immediate union was complete, with the exception of a very small chink in front, which has since contracted so much, that the point of a common probe can scarcely be passed through it.

Entertaining, as I do, a strong feeling that the results of an operation are more valuable tests of its efficiency than any reasoning, however plausible, I shall for the present be satisfied to leave the estimate of these proposals to the consideration of my professional brethren who may feel interested in the subject.

APPENDIX.

The following are the Cases referred to in the preceding paper.

Case 1.—Mr. D. P., ætat. 17, has a congenital fissure in the palate, extending through the soft velum. Swallows with facility, but articulates very imperfectly. The sound of the voice is very unpleasant, and many of his words are unintelli-

gible. The parts are not easily irritated by the fingers, and there is no disposition to vomit induced by touching the parts.

16th January 1844. The operation was performed to-day at one, P.M., according to the descriptions in the preceding pages. Three stitches were used. In the evening had a pint of thick gruel, as an enema. At noon, on the 17th, had the same quantity of beef tea, per anum, which was repeated in the evening, with the addition of a glassful of sherry. Same evening the anterior stitch was removed.

18th. At noon, and in the evening, had the beef tea and wine as before.

19th. Has swallowed a cup of beef tea this morning, and at noon the remaining stitches were removed. Wound has seemingly united throughout. A small portion of the posterior margin of the left side of the palate seems about to slough. Patient has been permitted to swallow beef tea at will, but has declined doing so, partly from pain and from the dread of causing the parts to separate.

20th. 9 A.M. The slough, as indicated yesterday, has separated. Union not so firm. At five, P.M., wound has opened to within half an inch of its anterior extremity.

29th. During the last eight days, the margins of the flaps have healed, and the union of the anterior part of the gap, to the extent of half an inch, has become firm. The patient left town this morning, with the intention of submitting to a second ope-

ration at a future period. The tone of his voice is slightly improved, but his articulation seems much as before.

March 1844. On the 19th of this month the edges of the gap were again pared, and brought together by three stitches. The treatment afterwards was the same as that above detailed. The wound united throughout, and in fifteen days the patient left town again, the parts being firmly united, with the exception of a very small chink in front, which was diminishing daily.

Six months afterwards Mr. P. had made no improvement in his speech, when he put himself under the tuition of Mr. Hunt, of Regent Street. In the course of a few weeks an extraordinary change was effected, and, ere long, the articulation was so different, that little more could be desired.

Case 2.—Miss W., ætat. 18, has congenital split palate, extending through the soft velum, and a fourth of an inch through the bones. Circumstances much the same as in the preceding case.

16th October 1844. Performed operation to-day, at 2 o'clock, P.M., as in the first proceeding in the case above detailed. In addition, however, the soft part of the palate was detached from the bones for the space of a quarter of an inch, or more, from the margin of the fissure towards the alveoli. Eight stitches were used, and the cut margins were very accurately adjusted.

19th. All the stitches removed to-day, and the wound has closed throughout. Patient permitted to swallow beef tea.

22nd. About half an inch of the anterior part of the wound has opened, the rest looking remarkably well.

In a few days more, as the margins of the little gap in front began to cicatrise, the aperture became less, and, after several applications of a heated iron, the aperture became so small that there was every prospect of its closing entirely.

In both of these cases, as soon as the incisions for the division of the levator muscles were completed, the soft parts were so much relaxed, that the gap was manifestly smaller than before; and during the subsequent steps of the operation, the flaps remained as flaccid as portions of skin on the margins of a wound. In the second operation on Mr. P., it was not deemed necessary to repeat the incisions above the flaps, as the levator muscles seemed, as yet, to have little or no influence. When this patient went to Mr. Hunt, the palate seemed little different from one which had been naturally well formed, excepting that there was no uvula, and the levator muscles seemed to have regained their power upon the velum.

In each case, the soft palate remained hard and somewhat thickened for months after the operation; and, on this account, the young lady (Case 2) has as yet made little progress in speaking. The tone of her voice, however, is much improved, and I have no doubt that the result will be as satisfactory as in the other instance.

ON THE

PULSATING TUMOURS OF BONE,

WITH THE

ACCOUNT OF A CASE IN WHICH A LIGATURE WAS PLACED AROUND THE COMMON ILIAC ARTERY.

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The recent occurrence of a case in St. Bartholomew's Hospital, of pulsating tumour originating in the walls of the pelvis in which a ligature was placed around the common iliac artery, has induced me to place before the Society, in connection with the narrative of this case, those circumstances in the history of the tumours of bone which bear on the important fact that certain of these tumours possess a pulsation identical in character with the pulsation of aneurism.

The pulsating tumours of bone differ from each other in their nature, they also remarkably differ in respect to the sources of their pulsation; and it will be chiefly in reference to the latter point of their history, that they will, in the following paper, be considered. Three distinct sources of pulsation in these tumours can be recognized: 1. The proximity of a large arterial trunk. 2. The development of blood-vessels and blood-cells, constituting a sort of erectile tissue within the tumour. 3. Enlarge-

ment of the arteries of the bone in which the tumour has originated.

Of pulsation in the tumour of bone dependent on the proximity of a large arterial trunk.—This is the most frequent source of pulsation in these tumours, of which the following are examples. Many years ago, two females were admitted into St. Bartholomew's Hospital, one middle aged, the other considerably older, with pulsating tumours in the upper arm. In each case the tumour occupied the whole circumference of the arm in its upper two thirds, and in each, it possessed throughout an equal and strong pulsation, which ceased on compressing the subclavian artery above the clavicle. In each case, the disease was considered to be an aneurism of the axillary artery. In the case of the younger patient, the ligature of the subclavian was recommended, but not assented to; she left the hospital, and lived several years afterwards. The other patient remained in the hospital to the time of her death. In both cases, I had the opportunity of examining the limbs; in the younger patient the disease was found to be an encephaloid tumour originating in the humerus, and covered above by the articular cartilage of the head of the bone. In the view of discovering if any unusual development of vessels within the tumour had produced its pulsation, I injected water into the posterior circumflex artery which penetrated the tumour, but the fluid escaped only at a few points from the surface of the tumour, and there were no large vessels distributed through

it. In the other case, the tumour originated in the humerus, and was composed of a firm, apparently gelatinous substance, about half an inch thick, and forming the walls of a large cavity filled by a serous fluid. It is not improbable that this was originally a solid cartilaginous tumour, in which the central softening and breaking up of its substance had taken place, to which such tumours are liable. In this instance, previous to the examination of the tumour, I injected the brachial artery with wax, and observed no remarkable distribution of vessels through the tumour: the injection passed very freely from the arteries into the veins of the limb. To this circumstance I am not disposed to attribute the pulsation of the tumour, as it is not unfrequently noticed in successful injections even of coarse wax into the arteries of the limbs, quite independently of disease. In both these cases the brachial artery was perfectly healthy, and, with its accompanying veins and nerves, was found closely united by cellular tissue to the tumour through its whole extent.

Subsequently to the foregoing cases, the following occurred in St. Bartholomew's Hospital. A man aged 30 was admitted under the care of Mr. Lawrence, with a swollen and painful state of the right knee-joint, consequent on a fall; for the removal of which, antiphlogistic treatment was successfully employed: but shortly afterwards, a painful swelling arose immediately above the knee, and gradually extended around the back part and sides of the lower third of the thigh. Near the tendon of the biceps,

a softening of the swelling indicated the probability of its containing matter, and accordingly a small incision was here made into it, from which about four ounces of arterial blood freely flowed. On examining the swelling more closely, pulsation in it was now discovered, and at the same time it was observed that the leg had become œdematous, and that the toes were colder than in the opposite limb. In consultation it was agreed that sufficient ground existed for believing the tumour to be a popliteal aneurism, and accordingly the femoral artery was tied in the middle of the thigh. The pulsation of the tumour immediately ceased, and its size gradually diminished; but after some time it again enlarged, became painful, and the skin covering it sloughed; the sloughing extended deeply into the tumour, but was unaccompanied by hæmorrhage. Whilst these changes were in progress, the man left the hospital, and shortly afterwards he sunk from exhaustion. I obtained permission to examine the limb, and found the tumour to consist of a compound of soft fibrous and dense osseous tissue, the latter situated deeply and extending around the femur, in which it appeared to have originated. The whole series of femoral, inguinal and lumbar absorbent glands were converted into osseous tumours. The femoral and popliteal vessels were sound. In the lower part of the thigh, the femoral artery was a little compressed and displaced by the ossified absorbent glands, which were closely united to it.

The specimens of disease from the three foregoing

cases are preserved in the Museum of St. Bartholomew's Hospital.

To the cases which have been related, I add three other examples of the same class of pulsating tumours, two of which were communicated to me by Mr. Hodgson of Birmingham, and the third by Mr. Lawrence. In Mr. Hodgson's cases, the disease was situated in the leg, and had originated in the lower half of the tibia, just above the inner ankle. In both cases the tumours were encephaloid in their structure; and in a communication recently received from Mr. Hodgson, he observes, "to what these tumours owed their pulsation I know not, but I thought it was derived from contiguous arteries." Mr. Lawrence's case is already recorded in the 17th volume of the Transactions of this Society: it was an instance of medullary tumour developed in the head of the tibia, attended at one period with pulsation and suppression of the pulse in the anterior and posterior tibial arteries at the ankle. In the account of the examination of the limb, Mr. Lawrence states that "the medullary tumour had protruded from the bone just at the division of the popliteal artery and the passage of the anterior tibial, through the inter-osseous ligament;" and he observes that "this circumstance accounts for the pulsation felt in the tumour at an early period; for the suppression of the pulse in the tibial arteries when the morbid growth was confined by the fascia of the leg; and for its subsequent return when the progress of the swelling through the fascia had liberated the arteries from pressure."

Here are six examples of pulsating tumours differing in their nature, and originating in different bones, but agreeing in the circumstance that no other source of pulsation was discoverable in them, than the contiguity of large arterial trunks; and to the same class of cases, the important one recorded by Mr. Guthrie appears to belong, in which a tumour about as large as an adult head, situated upon the right nates of a female, presented so decidedly the characters of aneurism, that it was believed to be so by Sir Astley Cooper, and other experienced surgeons who were consulted upon the case, and, accordingly, a ligature was placed around the common iliac artery. The following is an extract from a Clinical Lecture delivered by Mr. Guthrie on this case:—"I examined the tumour very carefully, and for some time I was very inclined to doubt whether it was a malignant tumour or an aneurism I had to treat; however, I at last came to the conclusion that it was the latter, firmly bound down by the glutæus maximus, which gave it considerable consistence, and rendered the pulsation less distinct, although it was decidedly manifest in every part; and on putting the ear to it, the whizzing sound attendant on the flowing of blood into an aneurism could be very distinctly heard; it was very audible previous to an operation being performed. The ligature of the common iliac artery was followed by a diminution of the tumour to the extent of one half, and the recovery from the operation was complete. Five months afterwards, the tumour again enlarged,

and she gradually sunk. On examination, the tumour was found to be composed of cerebriform substance. The arteries were healthy."*

Of pulsation in the tumour of bone, dependent on the development of blood-vessels and blood-cells constituting a sort of erectile tissue within it.—In the case of recent occurrence in St. Bartholomew's Hospital, the pulsating tumour originated in the ilium: it was soft, spongy and dark coloured, with cells dispersed through it, each about the size of a pea, and filled with blood. Bunches of convoluted vessels were drawn out of this spongy substance; and, when macerated, this substance was reduced to a tissue, closely resembling that of unravelled spleen or placenta. Here was a structure capable of enlargement by the distension of its vessels and cells; and assuming these to have been directly continuous with the arterial system, it may be added that the rush of blood into such a structure would give to the whole mass a pulsation resembling that of aneurism; at all events it is certain that this tumour did possess such a pulsation, which ceased directly the aorta was compressed through the abdominal parietes: moreover, that the tumour enlarged and became tense when the femoral artery below it was compressed, as an aneurism under similar circumstances would have done. I am indebted to Mr. John Lawrence, jun., Surgeon to the Sussex County Hospital, for the following case of pulsating

* London Medical and Surgical Journal. August 1834.

tumour, originating in the femur. A man aged 49, was admitted into the hospital with a small swelling in the right groin, under the femoral artery: it gradually increased to the size of an egg, and was then observed to pulsate, after which it rapidly increased. The pulsation continued, was uniform over the whole tumour, and accompanied by a distinct bruit. The limb became œdematous, and he gradually sunk with symptoms of disease in the lungs. The femoral artery was injected with size and vermilion. The tumour was found to have originated in the upper part of the femur: its immediate investments were some remains of the periosteum, with the surrounding muscles; interiorly it consisted of vessels intermixed with a soft gelatinous substance. The vessels formed more than half the tumour, were about the size of sewing thread, and very convoluted; that these vessels were directly continuous with the arterial system, was shown by the readiness with which they were filled by the coloured size injected into the femoral artery, and it may be inferred that the distension of these vessels with blood would be sufficient to occasion pulsation in the tumour.

Of pulsation in the tumour of bone dependent on the enlargement of the arteries of the osseous tissue.— Of this form of pulsating tumour, a few examples have been recorded. In one of these, related by Dupuytren, the disease originated in the tibia; and in the examination of the limb, the arteries ramifying around and penetrating the bone were found

much enlarged, and the veins of the limb were similarly altered.*

There are also cases recorded by Pelletan, in which the pulsating tumour appeared to have originated in the enlargement and rupture of the special nutrient artery of the medullary tissue;† and there is a case recorded by Morgagni, in which the foramen in the walls of the femur, transmitting this artery, had enlarged to the degree, that it easily admitted the little finger.‡ Mr. Luke, of the London Hospital, has furnished me with the following case, which apparently belongs to this class of pulsating tumours. A man, aged 20, suffered a fracture of the femur, which, at the end of seven weeks, was firmly united. A month afterwards, by a second accident, the bone was again broken at the same part. Re-union of the fracture ensued, but very slowly and unevenly. A tumour now formed in the situation of the injury: it was hard in some parts, soft and elastic in others. A grooved needle was introduced into it, and a jet of blood followed. The tumour increased to the size of a large melon, became very painful, and pulsation in it was now discovered. Suspicion of its being an aneurism in consequence arose, and in consultation it was determined to tie the femoral artery; this was done with the effect of stopping the pulsation of the tu-

* Leçons Orales, t. iv., p. 60.

† Clinique Chirurgicale, t. ii.

‡ On Diseases, Book IV. Letter II., art. 38, 39.

mour, and producing a diminution of it, to the extent of an inch in its circumference. About a month afterwards, the tumour again enlarged, but without the return of pulsation, and it was now deemed right to amputate the limb. The surface of the stump bled so profusely, that more than forty arteries required the ligature. The medullary artery was greatly enlarged, and threw out a forcible jet of blood. The man left the hospital with the stump healed, and in every respect well. On examining the limb, the lower third of the femur was found expanded into a spherical tumour, in the interior of which were cells of varying size, some of the largest about an inch in diameter, and they were filled with blood. The femoral and popliteal artery and vein were entire and healthy. From the diminution of the tumour, which immediately ensued upon the ligature of the femoral artery, it may be inferred that the arterial ramifications opened directly into the cells, of which the tumour was principally composed, and that in this manner the distension of the cells and pulsation of the tumour were produced; it may however be a question whether the general enlargement of the arteries of the thigh had accompanied the formation of the pulsating tumour, or had taken place subsequently, as a consequence of the current of blood through the femoral artery being obstructed by the ligature.

There is one circumstance in the history of these pulsating tumours which requires especial notice, as it appears to have a material influence on the

production of pulsation in them: this is the density and resistance of the structures which immediately invest them. In the instances of the development of the tumour within a bone, a thin osseous shell with thickened periosteum is its first and immediate investment; and in the instances of the development of the tumour upon the outer surface of a bone, thickened periosteum alone will be its first investment: moreover, according to the situation which the tumour happens to occupy, it will be more or less closely confined by the muscles immediately around it, which may then add considerably to the density and resistance of its coverings. Such were the circumstances of the case, recently in St. Bartholomew's Hospital, wherein the pulsating tumour, growing from both surfaces of the ilium, was in great part covered by the periosteum, much thickened, and it was, besides, closely confined by the iliacus internus muscle stretched over it on one side, and by the glutæus medius on the other. It may indeed be doubted whether any of these tumours would pulsate without the resistance derived in one or other direction from the bone or its coverings; and accordingly, in the case recorded by Mr. Lawrence, the medullary tumour developed in the head of the tibia pulsated in its early stage, but ceased to pulsate directly it had burst through the resistant coverings of the bone. A tumour originating in soft parts, unconnected with any bone, but situated close to a large artery, and confined within resisting structures, and thus approxi-

mating in its conditions to the pulsating tumour of bone, may, like it, pulsate in a manner to be mistaken for aneurism, of which the following is an instance.

A middle-aged man was admitted into St. Bartholomew's Hospital under the care of Mr. Earle, with a pulsating tumour, about the size of a small orange, situated immediately below the left clavicle, which presented all the characters of aneurism, and accordingly a ligature was placed around the subclavian artery upon the first rib. This was followed by an immediate cessation of pulsation in the tumour, and by a gradual diminution of it. The ligature separated at the usual period, and during the further progress of the case, no circumstance occurred to induce a suspicion of the disease being otherwise than aneurism; and, on the man's leaving the hospital, the tumour had diminished sufficiently to confirm this opinion of its nature. Six years afterwards, he was again admitted with general derangement of the health, from which he sunk. I injected the arteries of the upper half of the body with coarse wax, and found that the collateral circulation had been carried on chiefly by the inosculation of the supra and infra scapular arteries, which were greatly enlarged. From the first rib, the axillary artery, to the extent of about an inch and a half, was contracted and plugged by firmly adherent fibrin, but the coats of the artery were entire and healthy, and they presented no indication of having been the seat of aneurism. Immediately behind the

artery was a solid tumour of oblong form, about three inches in length and two inches in breadth. This tumour was moderately firm; internally it presented no other well-defined character than that there were cells dispersed through it, filled by a peculiar morbid deposit. A nerve, presumed to be the median, was closely connected with the tumour, and some of its filaments were expanded around the tumour in a manner to indicate it had originated within the sheath of the nerve. From this examination of the parts it appeared impossible to conclude otherwise than that there had been no aneurism, and that the tumour, originating within the sheath of the nerve, and closely embraced by the other nerves of the axillary plexus, had derived its pulsations from the axillary artery, close to which it was situated. This tumour is preserved in the Museum of St. Bartholomew's Hospital.

It is certain that there have been instances of the tumour of bone, the pulsations of which were, in every particular, identical with those of aneurism. In the case of pulsating tumour of the ilium, which will presently be related, the ear, resting against the walls of the abdomen, recognized in the tumour the strongly-marked bellows-sound of aneurism. So, likewise, in the case of encephaloid tumour of the pelvis recorded by Mr. Guthrie, it is stated "that on putting the ear to the tumour, the whizzing sound attendant on the flow of blood into an aneurism could be very distinctly heard." In the

instances of the development of blood-vessels and blood-cells in the tumour of bone, its pulsation, and the accompanying bellows-sound, may be caused by the rush of blood through these vessels and cells just as they result from the flow of blood through the sac of an aneurism. But in the instances where there was no other discoverable source of pulsation in the tumour of bone than the large artery situated close to it, yet the bellows-sound in the tumour was strongly marked, and could then be ascribed only to a change in the character of the pulsations of the artery dependent on the presence of the tumour. For true it is, as Dr. George Burrows remarked when engaged with me in the consideration of the case recently in the hospital, that in states of general anemia, also in diseases of the aortic valves allowing of regurgitation, limited portions, or even the whole, of the arterial system, without organic change in the vessels, may impart to the stethoscope a murmur approximating to the bellows-sound of aneurism. And if, under such circumstances, a tumour should happen to form in any region of the body so close to a large artery as to influence its pulsations, then, by the stethoscope, the bruit or bellows-sound may be recognized in the artery as forcibly as in an aneurism. Precisely this concurrence of circumstances happened in the case which will presently be related. The man had a tumour on the inside of his arm, in part covered by the biceps muscle, therefore directly over the brachial artery; and by the stethoscope, or by the ear alone,

the loudest bruit was recognized in the artery immediately above and below the tumour. These considerations show the little value to be attached to the existence of a bellows-sound in the diagnosis between aneurism and the pulsating tumour of bone.

I now proceed to relate the case of pulsating tumour of the ilium of recent occurrence in St. Bartholomew's Hospital, wherein a ligature was placed around the common iliac artery.

On Saturday the 18th of January, the patient was sent to the hospital by Mr. William Pennington. His age was 42, stature tall, and rather thin. During the last four years he had suffered at times from pain in the left hip-joint and back part of the pelvis, which was supposed to be from rheumatism and sciatica. His habits of life had been temperate; during the last twenty years he had been a butler in a gentleman's family, in the duties of which station he had continued to the day of his admission into the hospital. His complexion was sallow, appetite good, tongue clean, bowels regular, urine healthy, and nervous system tranquil. His pulse varied from 96 to 100. The sensation communicated to the finger placed upon any of the larger arteries was that of the vessel being præternaturally large, and of its beats being accompanied by a powerful vibration; through the stethoscope, placed on the brachial, or femoral artery, a distinct murmur or bruit was communicated to the ear. The pulsations of the left ventricle of the heart were loud, but not accompanied by bruit. By percussion, a

dullness of sound was recognized in the upper and front part of the right side of the chest.

On the inner side of the right upper arm, at about its middle, a tumour was discovered, about the size of a small orange, of an oblong form, and very loosely connected with the surrounding textures; its surface was smooth, it was wholly free from pain, and there was no pulsation in it. The man stated that he had first observed this tumour about ten years ago, and that during the last three years it had not increased.

The pulsating tumour was situated on the left side of the pelvis, its chief connection being with the ilium, in about the anterior half of its extent; it projected from both surfaces of the ilium, but more considerably from its abdominal surface, and it extended upwards to the crista. From the abdominal surface of the ilium the tumour extended downwards to Poupart's ligament, along its outer half, and into the cavity of the abdomen towards the mesian line. The tumour felt every where moderately firm, and when compressed it did not distinctly recede. The portion of the crista of the ilium which bounded the tumour superiorly, was uneven and thickened, and a little below the crista, near the anterior superior spine, a small moveable piece of bone was discovered, apparently involved in the tumour. Every where within reach of the fingers the tumour pulsated, not with a thrill or vibration, but with the deep heavy beat of aneurism. In the portion of the tumour projecting

from the dorsum of the ilium, the pulsation was less forcible than elsewhere. By the ear resting against the abdominal parietes, a bellows-sound in the tumour was plainly recognized. The external iliac artery could be traced by its pulsations for some way along the inner side of the tumour. Compression of the lower part of the aorta stopped the pulsation of the tumour. Compression of the femoral artery, below the tumour, produced an immediate enlargement and increased tension of it. A small artery was felt pulsating upon the lower and outer part of the tumour, its pulsations were vibratory, as in the rest of the arterial system, and were distinct from the deep, heavy pulsation of the tumour. The skin covering the outer part of the tumour presented a slight blush, and the man stated that the tumour was at the present time slowly increasing.

Such were the local features and the accompanying constitutional phenomena of the disease, respecting the nature and treatment of which we had to decide. In this decision, the following points were involved :—was this pulsating tumour an aneurism, and, if so, from what artery had it arisen; or was it one of the pulsating tumours of bone which have been shown in this paper to be not of very rare occurrence? In favour of its being an aneurism were the characters of its pulsation, with the accompanying bellows-sound, and its proximity to more than one artery sufficiently large to give rise to an aneurismal tumour of this mag-

nitude. Against its being an aneurism were the unevenness and thickening of the crista of the ilium forming the upper boundary of the tumour, also the existence of the moveable piece of bone just below the crista, and apparently involved in the tumour, inasmuch as both these circumstances belong to the ordinary progress and characters of tumours originating in bone rather than of aneurism.

Moreover, the situation of the tumour furnished an argument against its being an aneurism; for it might be urged that an aneurism, arising either from the external or internal iliac artery, would be more likely to extend in the direction of the abdominal cavity, than through the bones and thick muscles forming the walls of the pelvis. Much stress could not, however, be laid on this consideration, since an aneurism arising from the side of the external or internal iliac artery, which is in contact with the walls of the pelvis, might extend into the bone, just as an aneurism arising from the side of the thoracic, or abdominal portion of the aorta, in contact with the spine, does in some instances cause the absorption of the bodies of the vertebræ. Nor is it to be supposed that the tumour in the arm was out of our view; but its characters were so decidedly those of an innocent structure, that it appeared to have not the least bearing on the question of the nature of the pulsating tumour of the pelvis. In consultation, the preponderance of opinion was in favour of this

tumour being an aneurism, and the treatment was accordingly to be considered.

In the uncertainty respecting the origin of the supposed aneurism from the external or internal iliac artery, the decision would obviously be, that the common iliac should be tied. But from a careful consideration of the circumstances of the man's general condition, we agreed that so serious an operation could not be recommended, that its performance would be justifiable only under the circumstances of the patient being made thoroughly aware of his condition, and of his then desiring to take the chance of prolonging life which the operation afforded. His feeling was so decidedly expressed in favour of submitting to the operation, that I considered it my duty to undertake it.

I omit the details of the operation, with one exception. After the ligature of the iliac artery, the pulsation of the tumour completely stopped, when accidentally placing my fingers on its upper surface within the abdomen, I here felt an artery about the size of a crow-quill, pulsating forcibly, and with the vibratory character of the pulsations in the rest of the arterial system. At this discovery I was startled, for with full confidence in the power of the anastomosing circulation, I was not prepared to find, the instant after the ligature of the common iliac artery, that the blood was traversing the circumflex artery of the ilium with the same freedom and force as before the operation; the circumstance made me

apprehensive that the disease was something more than ordinary aneurism.

The operation was performed on Monday the 27th of January. Immediately after it, the patient took forty drops of laudanum in an ounce of camphor mixture. On visiting him in the evening, I found that he had been wholly free from uneasiness, and, to my surprise, that his pulse, which before the operation had varied from 95 to 100, was now only 80, and besides that, it was full and soft. This was satisfactory, as it permitted the hope that the character of the pulsations in the heart and arteries, noted before the operation, had for its cause excitement of the nervous system, and not, as had been apprehended, disease in the vessels. On the following morning, the pulse was accelerated, but no other change had occurred worthy of remark. Without any unfavourable symptom, the case proceeded to the middle of the second day, when the stomach became irritable, and the pulse extremely frequent and feeble, the abdomen swelled and became tender, there was almost constant vomiting, and with these symptoms he sunk, on the morning of the third day from the operation. With respect to the condition of the limb, I have simply to state, that no ill effect was produced upon it by the stoppage of the direct supply of its blood. At first the man stated that his foot was cold, and the rest of the limb numbed, and the sensibility of the skin of the leg was so far impaired, that slight friction of it was not felt. A bottle of warm water was kept in contact with the

foot, and the limb was enveloped in flannel. The numbness and coldness gradually diminished, and during the last twenty-four hours of his life, the man stated that the limb felt in no respect different from the other.

On examining the body, the effects of peritonitis were observed, but chiefly in the deeper parts and left side of the abdomen; here there was increased vascularity of the peritoneum, with the deposit of lymph on its free surface. Through the cellular tissue of the left lumbar and iliac regions, which was the tract of the wound, suppuration had taken place. The left common iliac artery was found to be only an inch and a half in length, and the ligature was placed around it exactly an inch below the bifurcation of the aorta. A loose conical clot was found within the artery, between the ligature and the aorta; and on detaching the ligature, the inner and middle coats of the artery were found completely divided. In the wall of the left ventricle of the heart, near its apex, there was a medullary tumour, about the size of a filbert. The free margins of the aortic and mitral valves were slightly thickened, and there were a few slight deposits of fatty matter in various parts of the aorta. Through the bronchial glands, medullary substance was diffused; and there were small deposits of the same kind in the lungs. Some slight remains of old tubercle were found in the upper part of the right lung.

On examining the tumour of the pelvis, it was

found that the portion of it projecting into the abdomen had displaced the external iliac artery from the walls of the pelvis, but that this artery had no other connection with the tumour than by loose cellular tissue. The internal iliac artery and its branches were in their natural situation, and wholly unconnected with the tumour. The abdominal surface of the tumour was covered by the iliacus internus muscle, and the portion of it projecting from the dorsum of the ilium was covered by the glutæus medius. Beneath these muscles, the periosteum of the ilium, much thickened, was extended over the tumour, excepting in its central and most prominent part, where the periosteum having been absorbed, the substance of the tumour was in contact with the contiguous muscles. I infer that the morbid growth originated in the periosteum and not in the bone, from the circumstance that it was intimately united to the periosteum, whilst it had no such adhesion to the bone, and from the appearance of the surface of the bone beneath the morbid growth, in which there were a multitude of minute holes, presenting the character of disease extending into bone rather than arising from it. It has been already stated that the substance of the tumour was composed of a spongy tissue with cells and convoluted vessels distributed through it. Through the two anterior thirds of the tumour the ilium was found unaltered, excepting the existence of the minute foramina in its surface, already noticed, but through its remaining third the integrity of the bone was destroyed, it had separated

into loose fragile fragments; consequently in this situation a direct communication existed between the two portions of the tumour on the opposite sides of the ilium.

I removed for examination the tumour from the inside of the arm; its connection with the surrounding textures was by loose cellular tissue, through which a congeries of large convoluted veins was distributed. A section being made of the tumour, its internal structure was, to our surprise, found to be identical with that of the tumour of the pelvis. It must be recollected that the tumour in the arm had never been painful, that its formation commenced ten years ago, and that during the last three years it had been stationary; yet with these evidences of an innocent structure, the tumour in question presented unequivocal features of malignancy, and thus furnished a striking illustration of the difficulty attendant on the diagnosis of tumours by their history and external characters.

The several specimens of disease from this case are preserved in the Museum of St. Bartholomew's Hospital.

P.S. Since the occurrence of the foregoing case, M. Roux has presented to the Royal Academy of Medicine in Paris, a memoir on pulsating tumours, designated aneurisms of bone, containing the following histories of this disease.*

* Bulletin de l'Academie Royale de Médecine, séance du Fevrier, 1845.

A tumour originated in the lower end of the radius of a man 36 years of age. On compressing it, fluctuation with a crackling sensation was communicated to the fingers. It was punctured, and arterial blood flowed from the opening. The tumour then rapidly enlarged and began to pulsate. The brachial artery was tied in the middle of the arm, upon which the tumour became flaccid, and ceased to pulsate; it remained stationary for five weeks, and then again increased with the return of pulsation. The arm was now amputated, and on the ninth day after the operation the patient died with symptoms of purulent deposits and phlebitis. The lower fourth of the radius had disappeared. The tendons of the wrist were raised and lodged in grooves on the surface of the tumour; its interiör was composed of cells filled with blood.

In a man 45 years of age, the head of the tibia enlarged with a projection from its outer side, the size of the closed hand. After the tumour had existed for several months, pulsation was discovered in it, but unaccompanied by bruit. The femoral artery was tied, the tumour immediately collapsed and ceased to beat, it gradually decreased, and ultimately disappeared, the head of the tibia completely recovering its form and size.

Other instances of pulsating tumour, originating in the tibia, have been recorded, wherein the femoral artery was tied, with the effect of arresting the progress of the disease; and in one instance, it is stated,

of completely curing it.* These, with the cases quoted from the Memoir of M. Roux, are to be regarded as examples of the variety of pulsating tumour of bone, wherein blood-vessels and blood-cells, constituting a sort of erectile tissue, are developed within the tumour. For the removal of such a simple vascular structure, the ligature of the principal artery of the limb may be effectual, but there must be difficulty in distinguishing this from the other forms of pulsating tumour originating in bone, and still greater difficulty in establishing a diagnosis between the pulsating tumour of bone and aneurism.

Two other cases of pulsating tumour, originating in bone, have been recorded, in which the main artery of the limb was tied, on the supposition that the disease was aneurism. One of these originated in the head of the humerus. On the 25th day after the ligature of the subclavian artery, profuse bleeding occurred from the wound, and the patient sunk. The tumour had diminished considerably, and it was found to consist of encephaloid substance.† The other case occurred in a soldier, nineteen years of age. As he was walking he felt a sudden pain in his left ham: on reaching home he found a swelling in the ham, which, in twenty-four hours, increased

* Leçons Orales, t. iv. Répertoire Général d'Anatomie, t. ii.

† Case of Medullary Sarcoma, engaging the upper portion of the os humeri, considered aneurismal, and for which the subclavian artery was tied above the clavicle. By John Nicol, M.D. Edinburgh Med. and Surg. Journal, July 1834.

to the size of a goose's egg, and it distinctly pulsated. The swelling increased, and the leg became œdematous. The femoral artery was tied, but the tumour continued to increase as rapidly as before the operation, and the patient sunk. The tumour was found to be firmly adherent to the periosteum of the femur and tibia. In its exterior were cysts containing a sanguineous fluid; its interior consisted in part of a fibrous tissue, disposed in lobes, and in part of a softer substance containing cells filled with serous fluid. The internal structure of the femur and tibia was healthy. The popliteal artery ran over the tumour and between two of the sacs of fluid on its surface, and through these it is stated that the pulsation had been communicated from the artery to the tumour. The right lung was converted into a brain-like mass.

In its mode of commencement, the disease in this instance presented so exactly the features of popliteal aneurism, that in the belief of its being so, the femoral artery was tied. The case is accordingly described as an instance of "excessively large fibrous tumour taken for an aneurism."*

* Observations in Pathology, by Dr. Kerst. Utrecht, 1839.

DESCRIPTION

OF A

MALFORMATION OF THE DUODENUM;

WITH NOTICES OF ANALOGOUS CASES.

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A MALE still-born infant from the lying-in ward of this Infirmary was examined by me on the 30th of April 1844. The body was eighteen inches long, and five pounds weight. There was dropsy of the abdomen and scrotum. The brain was natural, the cerebrum weighed twelve ounces, the cerebellum $\frac{3}{4}$ ounce, and the medulla oblongata 1 drachm. Chest:—both lungs sank in water, the right lung weighed 1 ounce, the left $\frac{3}{4}$ ounce; the heart weighed $\frac{3}{4}$ ounce, and the thymus body $2\frac{1}{2}$ drachms.

Abdomen:—the peritoneum contained an unusually large quantity of fluid; the stomach was natural, weight $1\frac{1}{2}$ drachm: the duodenum was much enlarged, and appeared like a bladder two thirds filled, and contained a greenish coloured fluid; the lower or most distant part from the stomach was imperforate, and of larger calibre than the upper part, so

that the malformed duodenum, as exhibited in the preparation, is somewhat of an oval shape, 6 inches long and 2 inches in diameter at the lowest and widest part.

The duodenum is completely closed by a transverse membrane at the lowest part; 2¼ inches above this, a valve is seen to extend across, nearly half closing the gut, proceeding from its concave side, and the central attachment of the septum being opposite to the mesentery or reflection of the peritoneum externally.

Around the membrane which closes the duodenum, the small intestine is attached, and when dried and distended with air is only about the thickness of a writing-quill. The peritoneum is continuous from the enlarged duodenum to the contracted portion of intestine adjoining, presenting a smooth unbroken surface, without any appearance of constriction.

The great intestines were unusually small, not fully developed; the colon contained some granular matter in round masses, like the dung of some rodents, of a light colour, in one or two places tinged with green. Part of it was sent to Dr. Lee.

The other abdominal organs were natural; the liver weighed 3½ oz., the kidneys ¾ oz., the supra renal capsules 1½ drachm, the pancreas 1 drachm.

Divisions in the intestinal canal have been most frequently met with where there have been other malformations. In acephalous monsters, for instance, a deficiency in the upper portion of the intes-

tinal canal is not uncommon, and has been observed by Otto, Klein, and others.

The œsophagus has been found terminating in a cul de sac, by Brodie and others. Schaefer* states, that a division between the stomach and duodenum has been observed four times, between the small and great intestine four times; that Pied found the duodenum receiving the bile-duct, and terminating in a cul de sac—and that a division in the small intestine, most frequently near its origin, had been found by seven other persons.

In the same essay, Schaefer relates a case which came under his own notice, of a child living some days with a malformation of the duodenum, very similar to the one which I have here described.

Billard† has noticed Schaefer's case, and another in which the child also lived some days, at the Foundling Hospital, Paris, with a similar malformation of the duodenum, which he has figured in his Atlas.‡ These were both male infants. The symptoms during life were, vomiting of a liquid resembling meconium, as well as all the ingesta: neither alvine dejections, nor urine, were passed by either of the infants. The whole of their organs, with the exception of the intestinal tube, were found to be healthy: there was an enlargement of the duodenum, and termination of it in a cul de sac—the portion of

* Journ. Complément. du Dict. des Sciences Méd., t. xxiv. p. 63.

† Traité des Malad. des Enfans, p. 347.

‡ Atlas d'Anat. Pathol., Pl. VI.

the intestinal canal below was unusually small, and contained only mucous matter.

The *post-mortem* appearances of these two male live-born children described in Billard's book, correspond, in most particulars, with those of the male still-born child here described: there is, however, a peculiarity to be seen in the duodenum of this one, not noticed in the others, which is, the membrane extending more than half way across, like a valve, at a distance above the obstruction.

From this circumstance, it would appear that there was a tendency to an increased development of the mucous membrane, and that the division and imperforation of the intestine below might be owing to a production of that membrane at an early period of fœtal life, extending across the intestine, and uniting with the mucous membrane of the opposite side. The obstruction so caused, by preventing the passage of the bile and pancreatic juices, would render unnecessary the full development of the intestines below the obstruction.

Simple enlargement of one of the valvulæ conniventes, without any surrounding disease, has been described by Baillie;* it was in the form of a projecting ring in the cavity of the jejunum, from which the canal of the gut was much narrowed: a similar case is mentioned by Houston.†

* Morbid Anatomy, 8th ed., p. 119.

† Descriptive Catalogue of Museum of College of Surgeons, Ireland, vol. ii., p. 44.

Meckel * states, that diminution in diameter "exists in several degrees; in the greatest degree it constitutes imperforation. It is always attended with the absence of a part, since on account of this defect, the part existing terminates in a cul de sac."

The œsophagus, duodenum and colon have been found divided into two canals.

There are numerous instances on record of varieties in the large intestines; the cæcum may be deficient, and also the vermiform appendix, and the rectum: the termination of the rectum in a cul de sac is by no means uncommon.

There have been monsters with only a more or less perfect abdomen, like the zoophytes. The intestines have never been wholly deficient, although it has happened that not the least rudiment of the cerebro-spinal axis, or heart, could be found. There never has been an instance of the head or thorax being formed separately.

The stomach has been found (according to Elben) while there was very little appearance of any other part of the intestinal canal. An instance is related by Andral,† in which the gastro-intestinal canal was reduced to the smallest dimensions, forming a straight tube which terminated in the rectum.

Among mammals, the great bat (*vespertilio noctula*, Linn.) has about the shortest intestines, which scarcely exceed the length of the animal's body.

* Manual of Descrip. and Pathol. Anat. (Eng. Trans.), vol. ii., p. 384.

† Pathol. Anat. (Eng. Trans.), vol. ii., p. 157.

The alimentary canal is partly formed, before there is any visible sign of spinal cord, brain, or heart: of the different parts of the alimentary canal, the one that is never deficient, is that which is first formed. According to Meckel and others, this canal is formed by the extension of the umbilical vesicle, which may be found outside the abdomen (the abdominal parietes are not formed till the second month), or along the cord in the form of a small tube, or else in the abdominal cavity forming a duct, and terminating at each end in a cul de sac.

The situation of the umbilical vesicle is about the lower third of the ileum, and according to the extension of the duct upwards or downwards will depend the extension of the small or great intestine. On the other hand, Wolff, one of the earliest observers, and Dœllinger, hold that the stomach and rectum are first formed, and that the canal proceeds from the two extremities, and unites at the ileum.

Tiedeman, Fleischmann, and others, are of opinion that the intestinal canal is divided into many parts, during the early periods of its evolution, and that these parts afterwards unite.

These opinions seem to be favoured in turns, some by the one and some by the other, of the various malformations.

More numerous and various malformations arise from an increase of the dimensions of the intestines than from the opposite state. Diverticula or appendices are frequent in the jejunum and ileum.

Monro* has divided these into two kinds—original malformations, and those formed consequent upon the protrusion of the intestines. Andral states that excrescences from the mucous membrane have been found in every part of the alimentary canal. An increase of thickness and density of the submucous tissue, giving rise to obstructions, &c., has been described by the same writer, and also by Houston, in the ileum, in his work before alluded to. Billard found in a child six days old, in a state of marasmus when born, a considerable thickening of the submucous cellular tissue at the end of the ileum and colon.

The comparative length of the alimentary canal, which is greatest in the mammalia, diminishes successively in birds, reptiles, and fishes, and, with certain modifications, is longer in graminivorous than in carnivorous animals.

It has been very much the practice to endeavour to find out something in the organization of the lower classes of animals analogous to the malformations in the human fœtus, but that this train of inquiry has thrown any light upon the subject is very doubtful. The present case, however, is not one where such assistance could be well called in, as it appears to me to be rather dependent on a pathological change *in utero*, and a stoppage of growth, than owing to any other remote cause.

* Morbid Anatomy of the Stomach, 2nd ed., p. 93.

ence at the first phalanx, and is in every respect properly formed. On the left hand, the thumb, index, and middle fingers are hypertrophied, the ring and little fingers remaining of the natural size, and presenting a curious appearance, in contrast with their gigantic neighbours. The finger most enlarged is the index, which measures five inches and a quarter in length, and four inches in circumference. The thumb and middle finger are hypertrophied in a less degree. The middle finger has a lateral incurvation outwards, occasioned apparently by a displacement of the extensor tendon, which forms a bridle along its outer edge. The other enlarged fingers are of the normal shape.

All parts of these hypertrophied fingers are equally developed in excess—the bones, articulations, integuments, and nails. The middle finger of the right hand and index finger of the left, which have attained the greatest growth, are fixed in an extended position, the girl being unable to bend them. The motions, however, of the several articulations are not destroyed, but the fingers are stiff, probably from long continuance in the straight position ; and the girl's inability to bend them appears to be owing to the flexor muscles in the fore-arm not having acquired a development corresponding to the fingers upon which they act, as the motions of those fingers which are hypertrophied in a less degree, are very little, if at all, impaired. The other parts of the upper extremities are in proper proportion to the size of the body.

There is merely a fulness at those parts of the hands from which the hypertrophied fingers proceed. Indeed, with these exceptions, all the different parts of the body are of appropriate size and form. The fingers generally feel cold and numb, but their sensation is not impaired, and she is not subject to chilblains in them. Pulsation can be detected in the digital arteries, but it is indistinct, which is not surprising, as the girl is in weak health, and the circulation is feeble. She constantly uses her hands in household work, and also in needle work, at which she is tolerably skilful.

On comparing the hypertrophied fingers of this girl, with those of the famous giant O'Byrne, whose skeleton, preserved in the Hunterian Museum, measures eight feet in height, I found that the two fingers in which the hypertrophy is most remarkable, are only half an inch shorter than the corresponding fingers of the giant.

I first saw this girl in the month of August 1839, at which time some wax models of the hands were made, and placed in the London Hospital Museum. On now comparing the hands with these models, I find that the middle finger of the right hand, and the fore finger of the left, have each grown an inch in length since that period.

This case is a remarkable example of partial hypertrophy, and though facts of this nature do not offer much of interest to the practitioner, they are worthy of record, and of the attention of the physiologist, as throwing light on the laws which regu-

late the development and formation of the body. There is, in this instance, an apparent absence of all those circumstances which seem favourable to excessive growth—a feeble constitution, sparing nutriment, no extraordinary exercise of the part, and no enlarged vessels or activity of circulation, and yet the hypertrophy so curiously confined to a few fingers is perfect in its kind: the common integuments, the nails, the bones and vessels, all have been uniformly and equally developed in excess. It would seem as if the formative powers which we see, in some few cases, exercised to excess in every part of the frame, so as to make a giant, had been limited in this instance to an insignificant part of the extremities.

My friend, Professor Owen has favoured me with the following communication:—

"College of Surgeons, January 6th, 1845.

"Having gone from the York Meeting last autumn, to the coal districts of Durham, in order to inspect the dwellings of the pitmen, I there saw a case of congenital hypertrophy of the fingers, analogous to the one of which you have shown me a drawing: the subject was a child, two years old, which the mother, wife of one of the pitmen of the great South Hetton Colliery, was nursing. Both hands were bandaged up, and she begged me to look at them: the middle finger of each, was nearly twice as long, and more than twice as thick, as the index finger, and the extremity of each was ulcerated, and additionally swollen by the inflammation;

but the mother assured me that the child was born with those fingers as much bigger than the others, as they were when I saw them, except that then they were sound, and that they had swollen at the ends since they became sore. It appears, therefore, that they had preserved their relatively disproportionate size since birth, by normal growth, with an additional and more recent increase, through the stimulus of inflammation.

"I noted the case at the time, merely as one of congenital excess of development, and, being fully occupied by other inquiries, sought for no further particulars."

I am indebted to Mr. Paget, of St. Bartholomew's Hospital, for the following particulars of a malformed hand, a cast of which was given to me by Mr. Dimond, of Frith Street, Soho Square. "It presents congenital hypertrophy of the first and second fingers of the right hand—the second is of enormous size. The cast is from a Spaniard, the Governor of the Fort at Luzon, one of the Philippine Islands. In 1837, when it was taken, he was about 50 years of age, and a well-made man in every part except his hand. At birth, the large fingers bore the same proportions as when the cast was made; but their extraordinary size produced no inconvenience: he could write very well, and he used his hand as if there were nothing unusual about it. To avoid observation, he had the habit of carrying his hand within the breast of his coat; but except from the curiosity it excited, he suffered no discomfort from it." On ex-

amining the cast, I particularly noticed that the middle finger, which was so largely hypertrophied, had the same kind of lateral incurvation outwards as was observed in one of the fingers of Eliza Hitchcock, so as completely to cross behind the adjoining fingers.

In the Museum of King's College, London, there is a cast of the left hand of an adult, in which the middle finger is hypertrophied, the others remaining of the natural size. The enlargement is uniform, and attended with a slight lateral inclination. The case is congenital, and the hands of other members of the family are reported to have been deformed in a similar manner.

This congenital form of hypertrophy is undoubtedly of rare occurrence, and there are very few cases of it on record. Mr. Power, of Dublin, has related an example which he met with in a girl five years old. He states, "the middle finger of her right hand is enlarged to the size it would naturally present in a very full grown, corpulent person: the metacarpal bone, and the phalanges, partake of this increased development, but are perfectly symmetrical. The thumb and the little finger are of the ordinary size, suitable to the age of the child, but the index and ring fingers are rather large, particularly the latter, and are divaricated from the middle, in consequence of its abnormal growth. On turning up the palm of the hand, the enlargement at the base of the phalanx of the hypertrophied finger is very remarkable, and gives this part of the hand the

appearance of that of a grown person. The integuments are natural, and her left hand, figure, and lower limbs are perfect and symmetrical." *

Dr. John Reid has published the particulars of a case of increased nutrition in the thumb and first finger of the left hand. The first exceeded the middle finger in length, by about half an inch, and had twice the circumference. The thumb of the same hand was about a quarter of an inch larger than that of the right, and had nearly double the bulk. The radial artery of the left, seemed to be double the size of that of the right arm. The temperature of the enlarged fingers was higher by a few degrees than those of the other hand. The difference in size existed at birth, and continued to increase till the boy's death, which happened when he was 13 years of age.†

In four of the foregoing cases, the hypertrophied fingers were bent to one side, and I suspect that in all of them this inclination was produced, as in the case of Eliza Hitchcock, by the tension of the displaced extensor tendon, which had not elongated in proportion to the increase in the size of the finger.

The deformity in these cases was of so offensive a character, that it is a point of some interest to determine whether it be possible by any mode of treatment to arrest the inordinate growth of the fingers in early life. It is questionable if this could be ac-

* Dublin Journ. of Med. Science, vol. xvii., p. 244.

† Lond. and Edin. Monthly Journ. of Med. Science, vol. for 1843, p. 198.

complished by any other means than by firm and long-continued pressure, the effect of which in preventing growth is well displayed in the atrophied feet and toes of the Chinese ladies. But in addition to the suffering to which this plan of treatment gives rise, the impairment of the functions of the part, caused by long-continued pressure, constitutes an insuperable objection to its employment. In a case where one finger only was enlarged to a great extent, and at the same time nearly useless, and interfering with the motions of the adjoining fingers, as was apparently the case with the Spanish Governor, the hypertrophied finger might be removed by operation. In other cases, the enlarged finger might be reduced to a more moderate size by amputation of the distal phalanx; and in this reduced state, the muscles would probably recover their power over it.

Excessive growth has been observed even less frequently in the toes than in the fingers. My colleague at the London Hospital, Dr. Little, lately showed me a cast of the foot of a child, in which the second and third toes were hypertrophied to double their natural size, and also united by the common integuments. Dr. Reid mentions an instance of increased nutrition in one toe of a female child aged two years. The middle toe of the left foot projected about three fourths of an inch beyond the great toe, and taking its breadth along with its length, it equalled in bulk all the remaining four toes.*

* Lib. cit.

ON THE

OPHTHALMIA OF PUERPERAL WOMEN.

By ROBERT LEE, M.D., F.R.S.,

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Received May 6th—Read June 24th, 1845.

In the history of the epidemic disease which prevailed in the lying-in wards of the Hôtel Dieu at Paris, during the years 1774 and 1775, it is stated by Monsieur Tenon that some of the puerperal women suffered from attacks of ophthalmia. From that period until the year 1825, it does not appear that any writer described inflammation of the eyes as a common or occasional occurrence in the diseases of puerperal women. Dr. Marshall Hall and Mr. Higginbottom then described in vol. xiii. of the Transactions of the Medico-Chirurgical Society, six "cases of destructive inflammation of the eye, and of suppurative inflammation of the integuments, occurring in the puerperal state, and apparently from constitutional causes." In all these cases the ophthalmia took place between the fifth and the eleventh day after delivery, and was characterized by redness of the conjunctiva, contraction of the pupil, intolerance of light, opacity of the cornea, and chemosis. The coats of the eye burst in two cases, and in these

before death took place the collapsed eyeball healed over. Inflammation and suppuration occurred in the integuments and cellular membrane of the superior and inferior extremities of four of these women. Dr. Hall and Mr. Higginbottom referred this fatal disease to a constitutional origin which they did not define. They did not examine the bodies of any of the individuals in whom they had observed the progress of the affection.

In Mr. Arnott's paper, "on the secondary effects of inflammation of the veins," published in vol. xv. of the Medico-Chirurgical Transactions, two fatal cases of phlebitis are related, in which a disease of the eye took place, similar to that described by Dr. Hall and Mr. Higginbottom. From the phenomena observed in these two cases, Mr. Arnott was led to infer that the inflammation of the eyes in puerperal women, was to be attributed to inflammation of the uterine veins: "considering the circumstances under which the affection of the eye took place," he observes, "its characters, and the deposition of pus under the integuments of the body, are we not justified (on comparing these with the known consequences of inflammation of veins, and the frequency of this affection in those of the uterus after parturition,) in attributing such disease of the eye to inflammation of the uterine veins, and to the introduction of pus into the system?"

In the same volume of the Medico-Chirurgical Transactions, p. 370, is recorded the history of a case which came under my observation in April 1829,

in which destructive inflammation of the eyes occurred in the progress of uterine phlebitis. Ten weeks after delivery, and six after the commencement of uterine and crural phlebitis, the conjunctiva of the right eye suddenly became so much swollen and inflamed that the eyelids could not be closed, and a copious secretion of an opaque fluid took place from their inner surface. The cornea soon became opaque, and the vision was entirely lost two weeks before death. There was little pain or intolerance of light. The left eye became similarly affected, without much pain, and both were so much swollen that they appeared to protrude from the orbits. The vena cava, internal and external iliac, and femoral veins, had all undergone the usual changes of structure which result from acute inflammation.

In 1832 another case came under my observation, which further contributed to prove that a relation exists between uterine phlebitis and the ophthalmia of puerperal women. In a patient of the British Lying-in Hospital, delivered on the 27th January 1832, obscure febrile symptoms took place, a few days after, without any pain in the region of the uterus. On the tenth day after delivery there was fever, with delirium, tremors of the muscles, and a peculiar dusky sallow complexion; the whole of the left lower extremity was swollen, hot, tense, and shining, and there was exquisite pain on pressure along the course of the iliac vessels on the left side, and down the inner part of the thigh. The conjunctivæ of both eyes suddenly became intensely red

and swollen, and the sight was much impaired, if not entirely lost. The right knee-joint became exquisitely painful, and a gangrenous spot appeared over the sacrum. Before death, with took place on the 18th February, the eyes had become enormously swollen, so that the eyelids could not be closed, and the vision was completely gone. The coats of the left common external iliac and femoral veins, deep and superficial, were all thickened, and their cavities plugged up with firm coagula. The same was the case with the epigastric vein, and circumflexa ilii. The glands in the vicinity of these veins were enlarged, red, and vascular, and closely adherent to the cellular membrane, and outer surface of the vessels. The vena cava, to a short distance above the entrance of the left common iliac vein, had its coats thickened, and a soft coagulum of lymph adhering to its inner surface. The uterine, vaginal, gluteal, and most of the other veins which form the left internal iliac, were gorged with pus, and lined with false membranes of a dark colour. The uterine branches of the right internal iliac vein were also filled with pus and lymph, but the inflammation had not extended beyond the entrance of the trunk of this vessel into the common iliac, and the right common and external iliac and femoral veins were all in a healthy condition. In the muscular coat of the cervix uteri, on the left side, was a cavity which contained about half an ounce of purulent fluid. The veins proceeding from this part of the cervix were filled with pus.

Many cases of uterine and crural phlebitis came under my observation, during the twelve years which intervened between 1832 and 1844; but in none of these did inflammation of the eyes take place, and an opportunity present itself of again proving by dissection, that the ophthalmia of puerperal women is the consequence of inflammation and suppuration of the uterine veins, or that there is a close relation between them. In the month of October last, the following case occurred, which appeared to establish, in the most conclusive manner, the truth of this view of the pathology of the affection, to confirm the accuracy of my former observations on uterine and crural phlebitis, and again to demonstrate, that in phlegmasia dolens, the inflammation commences in the uterine branches of the hypogastric veins, and subsequently extends from them into the iliac and femoral trunks of the affected side.

On the 10th of October 1844, Mrs. G—— was delivered with the forceps, after having been upwards of forty-eight hours in labour with her first child. For several days she appeared to be recovering in a satisfactory manner, when slight hæmorrhage from the uterus took place, with fever and constant difficulty of breathing. On the 6th of November, the dyspnœa and quickness of pulse having gradually increased without any pain in the region of the uterus, the vision of the right eye was suddenly lost. On the 7th, there was no intolerance of light, nor appearance of inflammation of the iris or conjunctiva. On examining the eye, on the 8th,

I found the iris covered with lymph, and there was a great effusion of clear serum under the conjunctiva, which was so much swollen that the eyelids could not be closed. The power of seeing with this eye was totally destroyed. There was severe pain in the right shoulder-joint, and in the right hand and fingers, which were stiff and swollen. The whole of the left lower extremity was affected with the swelling and other symptoms observed in phlegmasia dolens, and there were two large purple spots on the inner part of the left leg. On the 9th, there was no headache or delirium. The pulse was 130, and feeble, and there was much dyspnœa, without cough or expectoration. The pain continued in the right shoulder-joint, and the swelling and stiffness in the hand and fingers. The left lower extremity was swollen, hot, and colourless; and several more large dark purple spots, or blotches, had appeared over the inside of the leg, and one, the size of a crown-piece, over the right trochanter. There was no pain nor tension in the hypogastrium, or along the course of the iliac and femoral veins of the left side. The eye was in the same state.

On the 10th, the head was still perfectly clear, and there were no tremors or nervous symptoms of any kind. The conjunctiva of the right eye was red, the effusion of serum under it still greater; the iris was covered with lymph, and the bottom of the anterior chamber filled with it. There was merely a sense of stiffness experienced in the eye. Vision of the left eye was indistinct, the iris move-

able, and of a grey colour. Wine, ammonia, and beef tea were given freely, without much benefit. On the 12th the tongue was dry, and the pulse so rapid and feeble, that it could not be counted. Great prostration of strength and profuse perspiration took place, and a large gangrenous spot appeared over the sacrum; the swelling of the right hand had disappeared, the dyspnœa had increased, and there was occasional delirium. She died on the 16th, and I examined the body the following day. There was no trace of inflammation in any part of the peritoneal sac. The left internal iliac vein through its whole course was lined with a thin, false membrane, which closely adhered to the inner surface of the vessel, and near the uterus its cavity was filled with pus. All the veins on the left side of the os and cervix uteri, and those of the vagina, were inflamed and full of purulent fluid. The coats of the left common iliac vein were thickened, and its cavity filled with a soft, pultaceous mass, the outer surface of which firmly adhered to the vein. A coagulum of blood, three inches in length, was contained in the lower part of the vena cava, which had not been inflamed. The left common iliac vein, the superficial and deep femoral, and the saphena veins, were all filled with firm coagula of blood, but the coats of none of these vessels presented the alterations of structure usually observed after phlebitis. The cellular membrane of the whole of the left lower extremity was filled with serum.

In this case it was evident that the inflammation had commenced in the uterine and vaginal branches of the left internal iliac vein, and that it had extended from thence into the common iliac, and that the obliteration of the cavity of this vessel was the cause of the formation of coagula of blood in the external iliac, femoral, and saphena veins, and the consequent swelling of the left lower extremity. The rapid and destructive ophthalmia of puerperal women does not differ essentially from the violent inflammation of the lungs, cellular membrane, joints, and other parts so often observed to occur in cases of uterine phlebitis, and is undoubtedly referrible to the same cause. Ophthalmia, and all the secondary or remote symptoms which appear in the progress of phlebitis, have the same origin, and the rationale of all these symptoms must be the same. Some pathologists are inclined to believe that it is not so much to the condition of the nervous system, as to that of the blood and circulating fluids, however induced, that we are to look for an explanation of all the phenomena. That the blood becomes contaminated in cases of phlebitis, scarcely admits of doubt; but there is no direct evidence to prove that the pus formed in the veins actually mixes with the blood, and circulates through the system, and that the purulent fluid in the blood becomes arrested in some of the minute vessels of the eye, and there excites inflammation of the different textures of the organ.

ADDITIONAL OBSERVATIONS

ON

OBSTRUCTIONS OF THE PULMONARY ARTERIES.

By JAMES PAGET, F.R.C.S.,
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Received May 22nd—Read June 24th, 1845.

The following case of obstruction of the branches of the pulmonary artery by clots of blood, will, I hope, be deemed of interest sufficient to justify its admission, together with some remarks suggested by it, into the Transactions of the Society, as an appendix to the paper on the same subject, in the last volume. It presents the disease in a new and important form, making its progress unattended by alarming signs, and almost unobserved, and then destroying life with terrible abruptness.

A ruined wine-merchant, 55 years old, and who had drunk hard, had been in St. Bartholomew's Hospital, under the care of Mr. Stanley, since the 18th of April, suffering with stricture of the urethra and paralysis of the bladder. He was a bloated, unhealthy-looking man, and had occasionally slight œdema of the hands and feet; but he was so nearly well, that he never complained to any of his attend-

ants of more than the disorder of his urinary organs. Once, the house-surgeon, Mr. Parson, noticed that he had a cough; and he said that he was often subject to such an one, but thought it trivial. After his death, too, I learned from his fellow-patients that, in the last week of his life, he had sometimes spoken of having occasional shortness of breath, so that when he wished to sleep in the day, he was "apt to be taken with a panting;" and such an attack came on once or twice while he was walking in the ward. But these attacks were rare; they lasted only a short time, and they never appeared so serious as to elicit more than a passing remark from him; he mentioned them to none but the other patients in the ward; and within two hours of his death he talked in the same good spirits, in which he had been all day, of what he would do when he left the hospital with his stricture cured. On the morning of the 4th of May, the sister of the ward thought him looking unusually well. In the afternoon, after a short sleep, he proposed to a patient near him to have tea; but first he went to the water-closet, and, on his return, presenting nothing unusual in his appearance, he sat down on his bed-side. A minute after, he fell heavily on his face; and, with a slight distortion of his mouth and of one arm, died before two minutes more had elapsed.

The body was examined twenty-two hours after death.

The skull and brain presented nothing unna-

tural, except a few small earthy masses in the choroid plexuses. The cerebral substance had an ordinary appearance of vascularity.

The larynx, trachea and bronchi appeared healthy, their mucous membrane was pale. Some of the large bronchi contained a frothy mucus, slightly tinged with blood. The left pleural cavity was obliterated by general and rather close adhesions, the right was free, and contained a small quantity of fluid. The lungs were large and deep-coloured, but externally, appeared nearly healthy: their upper lobes were œdematous; the middle and lower lobes partially emphysematous, and containing a moderate amount of blood and serum. There was in no part of either lung a trace of pulmonary apoplexy, or of intense congestion. In the apex of the right lung was a slightly depressed cicatrix over a few dry tubercles, and a small healing cavity, full of thick tuberculous, and soft mortar-like substance.

The cavities of the right auricle and ventricle were large: their walls were of little, if at all, more than the natural thickness, but of firm and healthy texture. They contained a moderate quantity of fluid blood, with soft clots, and a mass of firm, colourless, semi-transparent coagulum, like a buffy coat, a prolongation from which, extended from the right auricle into the vena cava superior and some of its branches. The tricuspid and pulmonary valves were healthy, and so was the pulmonary artery, in the size and texture of both its trunk and all its visible branches. But nearly all the branches

beyond the primary divisions of the pulmonary artery contained clots of blood, which, from a comparison of them with those found in tied arteries, I judged to be from three to ten days old, those of the least age being in the largest branches. They were firm, cylindroid, and of various colours, black, brownish, pink, and yellow ochre, irregularly mixed. Some of them were much drier than recent clots, and grumous; others appeared invested and intersected by thin layers of white fibrine. Most of them filled the portion of the artery in which they lay, some did so to distension; a few of the driest and most decolorized adhered to the walls of the vessel, and left it rough and blood-stained when they were removed. The clots did not commonly extend continuously from any large branch of the pulmonary artery into many of its successively subordinate divisions; some of them were in the shape of thick cylinders, from half an inch to an inch in length, which filled a branch of the artery, but were not even in contact with the similar clots which filled adjacent branches; some extended from a larger branch into two or three smaller ones derived from it, filling them all, but ending abruptly; only a few branched many times, and filled a larger series of the branches of the artery. No branch of the pulmonary artery less than half a line in diameter appeared to contain any of these clots, and the pulmonary veins were healthy and empty.

The left auricle and ventricle were larger, and their walls were thicker, than in the healthy state.

They contained a small quantity of black fluid blood. The mitral valves were opaque, but pliant, and probably efficient. The aortic valves were in a similar condition, and of large size, adapted to the aorta itself, which was uniformly slightly dilated, and thickly beset with fatty, and a few earthy deposits in its coats. The large systemic arteries and veins were all healthy and empty, or containing a little fluid blood.

The whole digestive canal was healthy. Digestion appeared to have been active at the time of death, in both the stomach and the small intestines. The liver and pancreas were healthy: the spleen was of middle size, and soft.

In both kidneys, the pelvis and infundibula were dilated, the papillæ flattened and expanded, and the texture generally, but not uniformly, indurated and contracted, so that their external surface was lobed, and in parts coarsely granular. The ureters were dilated and tortuous, the bladder very large, and its walls thin and weak.

Behind the pharynx, near the right sterno-clavilar articulation, and in the course of the right femoral artery, there were small effusions of blood, like bruises, in the loose fibro-cellular tissue; but with these exceptions, no part of the body appeared as if peculiar or general congestion had existed.

Among the points suggested for consideration by this case, there are two of peculiar physiological interest, on each of which I beg to offer some remarks,

namely, the mode of death, and the probable cause of the coagulation of blood during life.

The case proves that, in certain circumstances, a great part of the pulmonary circulation may be arrested in the course of a week, (or a few days more or less,) without immediate danger to life, or any striking indication of what has happened. And the evidence of this case is confirmed by others of similar obstruction of the pulmonary arteries. This is, indeed, the only recorded instance of the disease being suddenly fatal in a person, who just before his death appeared in tolerable health; yet in some others it is certain that many of the pulmonary arteries must have been impervious long before the patient, though seriously ill, presented any distinct signs of such a hindrance to the circulation. For examples, among those whose cases are mentioned in my former paper, one* was suddenly seized with the signs of the obstruction during convalescence after a severe operation, and died in a few hours; in another,† the fatal attack came on and terminated as speedily, during convalescence after pneumonia; a third patient ‡ had long been greatly exhausted, but had no dyspnœa till shortly before her death; a

* Vol. xxvii., p. 167.

† Case from M. Baron, ibid., p. 164. [Since this was written, a somewhat similar case has been published by M. Richelot in the Gazette des Hopitaux, 29 Avril, 1845. The patient, a man 38 years old, appeared convalescent from pleuro-pneumonia, when suddenly he was attacked with a feeling of suffocation, and soon died.]

‡ Ibid., p. 182.

fourth* had various signs of dangerous import, resulting chiefly from obliterations of large systemic veins, but no dyspnœa till within an hour and a half of her death: yet in all these cases the characters of the clots by which the pulmonary arteries were obstructed, showed plainly that they had been for a week or more in progress of formation. Moreover, it may be observed that many if not all of those patients in whom the respiration was much affected during the formation and subsequent changes of the clots, had other extensive disease of the heart or lungs, to which the greater part of the dyspnœa might be ascribed.† It appears, therefore, that the obstruction of a large and quickly increasing portion of the pulmonary circulation, if it be not complicated by other disease, is usually unattended by disturbance of the respiration or any other important function.

Strange as this fact may at first seem, it may be explained: and its explanation will guide to that of the manner in which the increasing obstruction of the pulmonary arteries at last destroys life.

The absence of any constant deep distress of breathing during an extensive closure of the pulmonary arteries, shows that there can be no constant or

* M. Lediberder's case related by M. Baron in his "Recherches sur la coagulation du Sang dans l'Artére Pulmonaire," in the Archives Générales de Médecine, Paris, 1838, t. iii., p. 18.

† See the cases related in the former paper at pages 168, 171, 178, and a case by Dr. C. J. B. Williams, reported in the Medical Gazette, November 22, 1844, p. 257.

great congestion of those vessels of the lungs which are still able to convey blood; and the healthy manner in which, in some of the cases, the functions of other organs were discharged, makes it probable that all the systemic vessels were equally free from congestion. Now, for the avoidance of either general or pulmonary congestion, it is essential that equal quantities of blood shall in a given time pass through the systemic and pulmonary circulations respectively. To this end the conditions of the healthy state are exactly adapted; and in nearly all diseases of the respiratory and circulatory systems, a natural tendency to the preservation of the balance may be observed.

In the case of an obstruction of the pulmonary arteries—provided it is of small extent—the right ventricle will naturally increase the velocity of the blood, which it propels through the remaining open arteries; and it may so do this, and, at the same time, so dilate these vessels, that the quantity passing through the lungs in a given time shall not be diminished; in which case no change will be needed in the systemic circulation. But when many and considerable branches of the pulmonary artery are obliterated, and the usual quantity of blood cannot, in the usual time, by the aid of the compensations just mentioned, be transmitted through the lungs, then, the balance of the two circulations can be maintained, and congestion can be avoided, only by the movement of the blood through the systemic vessels being retarded, till the quantity traversing them in a given time is not greater than the reduced quantity which

now, in the same time, traverses the lungs.* This adjustment will be naturally effected—for, when the left ventricle receives, through those pulmonary arteries which remain open, a smaller than the usual quantity of blood at each dilatation, it must, in each contraction, discharge an equally small quantity; and the consequence of this (if other circumstances remain the same) must be a diminished velocity of the movement of the blood through the systemic vessels.

The peculiarity, therefore, of a person who has a considerable portion of his pulmonary arteries obstructed, must be, that, in direct proportion to the extent of the obstruction, his systemic circulation is less rapid, and his remaining pulmonary circulation more rapid, than before the obstruction took place. In such a state, it might be expected (and the cases prove) that the patient would live for a time without serious discomfort; for other facts show that an unusual rate of movement of the blood through a part, if unattended by distension of the vessels, does not immediately affect its function. Besides, although only a small quantity of oxygen may enter the blood, yet during rest a small quantity is sufficient for health; and there is no unusual retention of carbonic acid, for all the blood which enters the

* At least, this is the only way in which the balance of the circulations can be preserved, if, as in the recorded cases, neither the whole quantity of circulating blood, nor the quantity, whether moving or at rest, in the lungs, is materially reduced.

left side of the heart will, in the absence of any disease of the lungs themselves, have been sufficiently exposed to the atmosphere.

But such a state, though not necessarily one of discomfort, must always be one of danger; for if, after the circulation through those pulmonary arteries which remain open, has attained its maximum velocity, the circumstances which first led to the coagulation of the blood continue, then, as the obstruction by clots forming in the pulmonary vessels increases, so must the retardation of the blood in the systemic vessels. And this will go on till the blood ceases, or so nearly ceases, to flow in the systemic vessels, that the actions of the heart and nervous centres are no longer maintained; and thus the patient suddenly or slowly dies.

It thus appears probable, that the direct cause of death in these cases is not, as one might expect, in the state of the lungs, but in that of the brain or of the heart: the patient dies because these organs are not supplied with fresh blood. The mode of death is altogether peculiar. It is, in some respects, like death by asphyxia: in both, there is an arrest of blood at the lungs, but in this disease, the blood which does pass through them is aerated; in asphyxia it is not, and in it there are, therefore, all the signs of poisoning by carbonic acid, and the agony of want of breathing. Again, this mode of death resembles, in some points, death by anæmia; in both, the organs die for want of fresh supplies of

blood, but in anæmia there is no blood for them; in these cases, there is blood even in the organs, but it is not constantly renewed.

It accords with this explanation of the mode of death, that it is sometimes gradual, the movement of the systemic blood being gradually arrested; and that, sometimes, when the obstruction is very extensive, death ensues suddenly from an accidental event, such as unusual muscular exertion, which, by accelerating the movement of the venous blood, may discharge the systemic vessels more rapidly than they can be filled again through the remaining open pulmonary vessels.* Thus, the subject of this paper died directly after the exertion of defecation and walking; another was suddenly seized while walking, during convalescence.

In many cases, this mode of death is complicated, and accelerated by the effects of other diseases. When, together with the obstruction of the pulmonary arteries, patients have extensive bronchitis, or œdema, or other structural diseases of the lungs, as-

* Of the patients whose cases have been hitherto recorded, fourteen in number, six have died suddenly, namely, the subjects of M. Baron's 2nd, 3rd, and 4th cases; those of the 2nd case in my former paper (p. 167), and of this case; and a patient whose history is briefly given by Dr. Bright (Medical Reports, vol. i. p. 19); and to these may now be added M. Richelot's case,—making seven sudden deaths in fifteen cases. In some of the others, sudden attacks, or great aggravations of dyspnœa, or sinking, such as in a rather worse degree might have proved suddenly fatal, have been observed: see especially the 5th case in my former paper, p. 174.

phyxia may add its signs, and quite overwhelm those of the ceasing movement of the blood. Several of the cases show this very plainly; and others show, that when the lungs are in their own texture healthy, death, whether gradual or sudden, is tranquil.

With respect to the second point proposed for consideration—the cause of the coagulation of the blood in the pulmonary arteries—if all the recorded cases of this disease be brought together, there will be found great diversity in the circumstances leading to the formation of the clots.

In some cases, it appears almost certain that the chief reason why the blood coagulates is, that its passage through the small vessels is arrested by disease of the left side of the heart, or by pneumonia, pulmonary apoplexy, or some other disease external to themselves obstructing them,* or by some morbid substance collecting in them.†

In other cases, the coagulation may be chiefly ascribed to the blood almost ceasing to be moved by the weakened hearts of those who, by long-continued disease of any kind, have become utterly exhausted ;‡ a condition which, like the preceding, will also greatly increase the chances of coagulation taking place, when it coincides with any other of the circumstances in which it may happen.

* Such are M. Baron's 2nd case, p. 17; those referred to in the former paper, pp. 163, 168, 171, 173; Dr. Williams's case.

† As in the three cases in the former paper, pp. 165, 166.

‡ Such, perhaps, as the patient in the 6th case, in the former paper, p. 182, and M. Baron's 3rd case, l. c., p. 9.

Again, in other cases the blood coagulates in inflamed pulmonary arteries, as it does in inflamed veins;* and, probably, it would coagulate in any pulmonary artery whose coats were much roughened by disease.†

But in the case just recorded, and in at least four others,‡ the blood appears to have coagulated, independent of all these conditions; the texture of the lungs was, if at all, only trivially diseased; the coats of the obstructed pulmonary arteries were healthy, the heart has appeared sufficiently powerful, and the patients have shown no signs of extreme exhaustion before, or while the clots were forming. In these mysterious, but most interesting cases, therefore, we must look for explanation to the condition of the blood itself.

It is very important to observe that in all these cases, though there was no manifest mechanical hindrance to the movement of the blood, the clots were in masses, not laminated; indicating that the blood, when it coagulated, must have been at rest. And that it was at rest, both during and after coagulation, is further evidenced by the older clots in this

* Cruveilhier, Anatomie Pathol., Livr. xi., Pl. 2, 3; and others.

† In M. Baron's 4th case, l. c., p. 22, this may have been the cause of the coagulation.

‡ M. Baron's 1st case, l. c., p. 6; Dr. Bright's case, l. c.; and the 4th and 5th cases in my former paper, pp. 174, 178. (Probably, also, M. Richelot's case, already cited, may be added to these, for the texture of the lungs is not said to have been materially changed.)

and a former case being in numerous pieces, not branched and extending continuously through all the branches of the arteries in which they were found; for this must be ascribed to the breaking of a large continuous clot, (such as could only be formed during rest,) in the act of contraction which succeeded to the simultaneous coagulation of the whole mass. Now, rest being a sufficient condition for the coagulation of ordinary blood, we need not suppose that, in these cases, there was such a morbid state of the blood as rendered it peculiarly coagulable; especially when such a supposition is contradicted by its fluid, or softly coagulated state, in all the vessels except the pulmonary arteries. Neither can it be supposed that the blood ceased to be moved or coagulated because portions of it died; for, besides that it is probable that coagulation itself is partial organization, there is evidence that, in one of the cases,* the blood after coagulation became more highly organized.

In the absence, then, of all evidence of a peculiar disposition of the blood to coagulate, and in the assurance that the coagulation took place during the rest of the blood, although no cause of obstruction external to the vessels could be discerned, I will suggest, that the chief cause of the stoppage of the blood, and of its consequent coagulation, in these cases, is some morbid state affecting its constitution, so as to increase that adhesion of it to

* Case V., and Pl. III. fig. 3, in the former paper.

the walls of the vessels which constitutes, even in the healthy state, the greatest resistance which the heart's power has to overcome.

In this view, the frequent coincidence of granular degeneration of the kidneys with old clots in the pulmonary arteries is interesting.* Many of the signs of that disease of the kidneys, and especially the enlarged heart, and the dilated and tortuous arteries so often found, indicate that there is an unnatural difficulty in the transmission of the blood through the capillaries or the small arteries and veins. And to explain this difficulty, it is not unreasonable to suppose that the diseased blood adheres more than healthy blood does to the walls of the vessels, the urea or some other retained constituent of the urine acting in the same manner as experiments † have shown that salts of baryta and some other bases will act, which, when introduced in even very small quantities into the blood, hinder, or altogether prevent its passage through the small vessels, although the heart be acting with full force.

On this hypothesis, all the circumstances of these cases may be explained. At first, we may suppose

* The coincidence existed in at least three of these five cases, viz.: in this present case, in Dr. Bright's, and in Case V. p. 178, in the former paper: in the other two cases, the state of the kidneys is not mentioned. They were granular in Cases II. and III. in that paper, and may have contributed to the coagulation of the blood, although the state of the heart and lungs may have been an equally or more favourable condition for that result.

† Especially those of Mr. James Blake.

all the blood moving at an equally retarded rate through the pulmonary capillaries. At length some of it ceasing to move in a part where an accidental congestion, however slight, makes its passage more peculiarly difficult, clots will be formed in the corresponding pulmonary arteries, to which result also, the absence of anastomoses between the larger branches will contribute. After the obstruction of a part of the pulmonary circulation, a larger portion of the force of the right ventricle will be available for the rest: this, therefore, may go on at least as well as before, till, the evil condition of the blood continuing, it ceases to move in some other part, and fresh clots form. Thus the disease may make progress till, in either of the ways already spoken of, death ensues, or, as has happened in at least one case,* the blood may regain a healthier state, and the obstruction and coagulation ceasing, the clots already formed, acquire higher organisation, and, adhering to only one part of the arteries which they filled, may allow blood again to flow through them: thus the patient may recover.

In conclusion, I will venture to suggest that many cases of sudden death, for which no cause has been found, or which have been ascribed to some insufficient or improbable cause, have depended on clots obstructing the pulmonary arteries; and that the clots of the same kind, which are often found in the systemic veins, and are usually ascribed to

* Case V. p. 178, in the former paper.

phlebitis, though the coats of the veins are healthy, are the consequence of stoppage of the blood similar to this which I have described, and from a similar cause. It is no argument against the supposition of the frequency of such arrests or slow movements of the blood that the pulse continues: for the pulse is not, necessarily, an indication that the blood is flowing through the vessels, any more than a wave is sure evidence of a tide.

P.S. Since the preceding paper was read to the Society, I have met with the observations of my esteemed friend Dr. Angelo Dubini on the same subject. These are contained in an essay full of good practical pathology, which was read to the medical section of the Scientific Congress at Milan, in September 1844, and are published with an appendix containing a translation of the abstract of my former paper, in the "Annali Universali di Medicina, di Febbrajo, 1845."

Dr. Dubini states that he has been in the habit of examining clots in the branches of the pulmonary arteries since 1839, when his attention was first called to them by a memoir which was read by Dr. Alessandro Gambarini at a monthly meeting at the Milan Hospital.* He says that my observations are in almost perfect agreement with his own and Dr. Gambarini's; but I regret that he has given so few particulars of his cases, that the

* Dr. Dubini does not refer to this memoir as one published, neither can I find it in any of the Journals of 1839 or 1840.

following are the only matters of fact directly bearing on the subject, which I can gather from his own essay, and his commentaries on mine.

He has found in "surprising frequency," clots obstructing the branches of the pulmonary arteries, and presenting all the characters of clots formed in vessels at various periods before death. In a series of twelve cases, the lungs in eight presented no such change in their proper texture as could give rise to obstruction of the capillary circulation. "In some of them there was slight pulmonary œdema: in others some engorgement by bloody serum; in others the texture of the lungs was soft and dry; in one there were clots in the arteries of one lung which was not œdematous, while the other, which was œdematous, contained none. Of the four cases in which the pulmonary texture was in some degree affected, two had circumscribed or lobular hepatization with gangrenous excavations, the two others had phthisis with tuberculous cavities. In most cases the clots occupied the large divisions of the pulmonary artery, while the trunk and secondary branches were free."

Pulmonary emphysema, organic disease of the heart, spots of yellow deposit in the inner coat of the pulmonary arteries, and dilatation of the branches thus diseased, were frequent concomitants of the obstructions; but no similar clots were found in cases of hepatization.

In endeavouring to explain the formation of clots in the pulmonary arteries, Dr. Dubini rejects the supposition of an inflammation (like phlebitis) of

the obstructed vessels, as an improbability. He suggests that the cause of the coagulation during life is in the altered crasis of the blood; but he thinks that there are many facts in opposition to the theory, " which destroys every hope of success in the explanation of the genesis of the clots."

TWO CASES

OF

ANÆSTHESIA AND LOSS OF MOTORY FUNCTION OF THE FIFTH NERVE.

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Received June 2nd,—Read June 24th, 1845.

An especial interest is imparted to the diseases of the fifth cerebral nerve, by the intimate connection which subsists between it and the senses of sight, smell, and taste: and as the very mechanism of one of these senses, namely, taste, is still a subject of dispute among physiologists, I trust that a report of two cases of anæsthesia of the fifth nerve, which have lately been under my notice, may prove not uninteresting to the present meeting.

Serres* claims to have been the first who observed and described anæsthesia of the fifth nerve; but, in fact, Herbert Mayo† had anticipated him by a year. Indeed, Bellingeri,‡ in 1818, had given some

* Serres, Anatomie comparée du Cerveau. Paris, 1824-6. Vol. ii., p. 67.

† Mayo, Anatomical and Physiological Commentaries. London, 1823. No. 2, p. 12.

‡ Bellingeri, Dissertatio Inauguralis, &c. Aug. Taur., 1818.

account of a disease of the sensitive portion of this nerve, and had made several experiments on his patient, as to the effects produced by it upon the senses of taste and smell. A body had been dissected in 1809 by Fribault and Maréchal, in which the fifth nerve was found almost destroyed by a tumour, originating from the dura mater: but this case is less interesting than those by Mayo and Serres, on account of the very scanty history which is given of the phænomena observed during life, neuralgia being the only symptom recorded. It seems strange, however, that this dissection of Fribault and Maréchal's (which was published in the *Journal de Médecine* for 1812) should not have attracted more attention, and have convinced medical men that the seat of neuralgia of the face was to be sought in the fifth, and not in the facial nerve, which in those days was so frequently divided, in the hope of curing the disease.

Since the publication of Mayo's and Serres's observations, many cases of anæsthesia of the fifth nerve have been noticed, and their various symptoms and *post mortem* appearances detailed, either in systematic treatises, or in the pages of medical journals. I have collected the particulars of forty-seven of these cases, many of which are of great interest, as illustrating the functions of the organs of sense.*

* In arranging these histories, I have been struck by the much greater frequency of disease affecting the fifth nerve on the

Vivisection (which, in the hands of Magendie and others, has been of such important service in explaining the phænomena of motion and feeling) has thrown but little light on the physiology of smell and taste: these are too delicate and purely subjective to admit of being profitably studied, except on the human body. Even then, *positive* conclusions can be drawn only where a careful *post mortem* dissection has shown what nerves, or what portions of the brain, have been diseased, and to what extent the morbid process has affected their structure.

left, than on the *right* side of the body. Out of forty-seven recorded cases of the kind, thirty occurred on the left, and only twelve on the right; whilst in five patients, *both* nerves were affected at the same time.

Published cases of Anæsthesia and loss of motory function of the 5th Nerve.

Right side.	Both sides.	Left side.
Bellingeri, 1. Serres, 1.	Jodin, 1.	Mayo, 1. Bell, 4.
Bell, 1. Watson, 1. Gama, 1.	Nélaton, 1.	C. W. Bell, 1. Stanley, 1.
Carron du Villards, 1.	Cruveilhier, 1.*	Abercrombie, 1. Horner, 1.
Boyer, 1. Bishop, 1.	———, 1.†	Colles, 1. Whiting, 1.
Stamm, 1. James, 1.	Annan, 1.	Bishop, 1. Noble, 2.
Tavignot, 1. Montault, 1.		Carré, 1. Montault, 1.
		Voisin, 1.
		Tanquerel-Desplanches, 1.
		Landouzy, 1. Romberg, 4.
		Corrigan, 1. Stanski, 1.
		Jobert, 1. Vogt, 1.
		Magendie, 1. Tavignot, 1
		Salomonsen, 1.
		Add the cases related in the present paper, 2.

* Taste and smell were lost in this case on both sides, but the tumour involved the fifth nerve on the *left* side only.

† The author's name is not given in the *Gazette Médicale*, where the case is copied from the *Bullettino delle Scienze Mediche*.

Magendie's* well-known experiments have shown that slough or ulceration of the cornea, and loss of the humours of the eye, invariably take place whenever the fifth nerve is completely divided within the cavity of the skull in a living animal. The morbid phænomena observed in the human subject have been, in many cases, at seeming variance with the deductions from these experiments. A patient has had, for instance, total loss of feeling in all the parts supplied by the fifth nerve—in the skin, the conjunctiva, the tongue, the lining membrane of the nostril; and yet the eye has for many months remained free from inflammation, its coats and humours retaining a perfectly healthy appearance. In other cases, again, the eye has undergone that destructive process which invariably follows the section of the nerve in the experiments of Magendie.

It becomes, then, an object of much practical interest to determine the causes of such different terminations, in apparently the same disease.

Sir Charles Bell thought that, inasmuch as this anæsthesia was frequently accompanied by palsy of the facial nerve, the constant exposure of the conjunctiva to floating particles of dust and other foreign bodies produced, at first, inflammation, and, at a later stage, slough of the cornea. But this theory is overthrown by the fact, that, in several

* Magendie, (et Desmoulins) Anatomie des Systèmes des Animaux à Vertèbres. Paris, 1825. 1st part, pp. 710 et seqq. Leçons sur les Fonctions et les Maladies du Système Nerveux. Paris, 1841.

recorded instances of destructive inflammation of the eye, there was at the same time palsy of the third cerebral nerve; so that the upper lid constantly hung over, and protected the cornea: and again, we every day see patients who have their *orbiculares palpebrarum* muscles paralysed, in consequence of disease of the seventh nerve, without their becoming subject to any inflammation of the eye in consequence.

Others have asserted, that the eye is endangered or not, in proportion as the disease has, or has not, implicated the Casserian ganglion. But the records of *post mortem* dissections prove this theory likewise to be untenable. In a case, related by Bell,* where the ophthalmic branch of the fifth nerve was diseased, slough of the cornea took place; but in another case, by Bishop,† where all the three foramina for transmitting the divisions of the fifth nerve were obliterated, the cornea remained sound.

Destruction of the trunk of the nerve between the brain and the Casserian ganglion, or degeneration of the latter, was followed by slough of the cornea in three patients, examined by Abercrombie,‡ Stanley,§ and Horner.‖ On the other hand,

* Bell, The Nervous System of the Human Body. 3rd edit., 1836, p. 271.

† Paper read at the Royal Society; Medical Gazette, 1833, vol. xiii., p. 463.

‡ Abercrombie, Diseases of the Brain and Spinal Cord. 3rd edit., 1834, p. 424.

§ Med. Gazette, vol. i., p. 531.

‖ American Journal of the Medical Sciences, 1829, vol. iv., p. 58.

Serres,* Whiting,† and Gama,‡ found the same morbid changes in three other patients, and yet in no one of these cases did slough of the cornea ensue; and in only one of them was there any inflammation of the eye at all.

The cases which I am about to relate to the Society exemplify these two forms of diseased action. In the first patient, anæsthesia of the fifth nerve has existed for sixteen months; and yet there is not the slightest inflammation of the eye, nor any opacity of its humours. In the other case, inflammation attacked the eye within eight months from the commencement of the disease in the nerve; the cornea became opaque; lymph was effused into the pupil and anterior chamber; and when I last saw the patient, there was every probability of a total disorganization of the globe.

Case 1.—Sophia Donald, aged 53, residing at Beckenham, applied at the Ophthalmic Hospital, (February 2nd, 1844,) complaining of general dimness of sight, and double vision. The appearance of the eyes was healthy, except for some sluggishness in the motions of the left iris; the pupillary opening of which was larger than that of the right. There was no strabismus, nor any irregular action of the muscles of the eye-ball. Her pulse was natural. Her bowels were confined; but in other respects her health was pretty good.

* Serres, loc. cit. † Case quoted in Bell's Work, p. 352.

‡ Gama, Traité des Plaies de la Tête, &c. Paris, 1830, p. 180.

She stated that the dimness of sight in the left eye began about two months ago ; and was followed, in about three weeks, by dimness in the other. Violent headache, which was almost constant during a month, preceded the impairment of vision. This headache was so severe (her friends told us) as, at times, to affect her reason. In the course of conversation she incidentally mentioned that one side of her face "felt dead." This led to a further examination ; and it was found that the fifth nerve on the left side was completely paralysed. Before detailing the results of experiments, I may mention that after a few doses of purgative medicine, the double vision and the dimness in the right eye entirely ceased ; the general dimness in the left eye still remaining.

For the sake of comparison, I have arranged the results of Magendie's experiments and the symptoms observed in this patient's case in opposite columns ; and it will be seen that they correspond in every particular, except as regards the state of the eye and vision.

*Results of the division of the fifth nerve according to Magendie.**

Results of experiments on patient.
Feb. 9, 1844.

1. Muscles of mastication cease to act.

The difference between the two masseter muscles is not so evident as that between the temporals; on account of the movements of the coronoid process of the jaw interfering with the examination. The hands being placed on both temporal muscles, at each elevation of the jaw the muscle of the right side contracts, the left remains motionless. By putting a finger into the patient's mouth and applying the thumb so as to grasp the masseter, the flabbiness of the left muscle as compared with the right is very marked.

2. All sensation in the face lost: the muscles retaining their power.

Skin.—She cannot feel the prick of a pin on any part of the left half of the face; except on a small space (bounded by the zygoma, the course of the facial artery, and the lower margin of the jaw-bone,) supplied by twigs from the cervical plexus. The skin of the temple is insensible, as far back as a line drawn from the tragus vertically upwards. The whole of the pinna retains its sensibility.

Eye.—The ocular and palpebral conjunctiva is utterly without feeling. It did not redden when scratched with a pin, or pinched with forceps; nor did any acrid substances, as snuff, nitrate of silver, or sulphate of copper, induce injection of its vessels.

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* Loc. cit.

Nose.—A feather may be thrust into the left nostril and turned in every direc tion without being felt. The Schneiderian membrane on this side is dry, while that of the right nostril secretes naturally.

Tongue.—The patient cannot feel the prick of a pin or the pinch of a forceps on the left half of the tongue; and in eating, her food collects between that part and the cheek.

3. Taste lost by the anterior part of the tongue: remaining at the base.

Quinine, introduced on the tip of the finger and rubbed upon the *base* of the tongue on the left side, was tasted: but it was not tasted when rubbed upon the *edge* of the tongue towards its *fore part*.

4. Smell lost.

When the right nostril is closed, she cannot smell essence of bergamot, rhubarb, kreosote, or snuff; which she does perfectly with the right nostril open. Vapour of ammonia snuffed up the left nostril produced but slight effect—no more than might be caused by some passing into the right nostril by the posterior opening: but when drawn into the right nostril, it took away her breath, and produced a flow of tears from the right eye, but none from the left.

5. Hearing lost.

With the right ear she can hear my watch tick at a distance of nine inches. With the left she cannot hear it except when placed almost in contact with the ear. (Her hearing, she says, was formerly as good on one side as on the other.)

6. Division of the fifth nerve in rabbits, guinea-pigs, cats, and dogs, produced—

i. Blindness.

The patient could still distinguish, at this stage of the disease, the form and colour of large objects with the left eye; so that the sight on the affected side was only *impaired*, while the senses of smell and taste were *totally lost*. A year later (1845) amaurosis of the left eye was complete; but then this was accompanied by paralysis of part of the third nerve, and the disease had probably implicated the second also.

ii. Immobility of the iris: the pupil sometimes dilated, sometimes contracted.

The left iris, when exposed to the light, contracts; slowly, but as completely as the right. [As the disease went on, this power of contraction was lost—1845.]

iii. Insensibility of eye-ball.

See Exp. 2, *ante*.

iv. Suppression of tears: the eye still remaining moist from mucous secretion.

The patient told us, of her own accord, that she had yesterday some little dispute with her husband, and cried; when she found, to her surprise, that she could weep only with the right eye.

The conjunctiva still remains moist.

v. Cessation of winking; except when the eye was exposed to

The orbicularis palpebrarum, and indeed all the muscles supplied by the facial nerve, are quite perfect in their actions.

Winking is performed simultaneously on

strong sunlight.

both sides; and the introduction of a foreign body, such as a pellet of paper, between the left globe and the palpebræ, not being felt, does not induce any increased action of the orbicular muscle.

On the right side the fifth nerve is quite unaffected, either in its motory or sensitive portions.

After attending two or three times, the patient was lost sight of, until October 3rd, when I met her accidentally.

A considerable change had taken place in her condition. There was complete *ptosis* of the left upper eye-lid; and when this was raised with the finger, she had no perception of light. When placed, however, in the full glare of sunshine (the right eye being shut), she said she could perceive "some little glimmer." The left iris was quite motionless, even when the sun's rays fell directly on the retina. Contractions of the iris did now and then take place, but they were irregular, and quite independent of light. Besides the *levator palpebræ*, the *superior rectus* muscle was paralysed: the eyeball could be freely moved downwards, or inwards, or outwards; but not upwards. The humours and coats of the eye were perfectly clear and healthy-looking; although the disease of the fifth nerve had now been going on for about ten months.

The patient stated, that from February (when she ceased to attend at the Ophthalmic Hospital) until the last week in July, no particular change took

place in her health. At the latter period, pain came on in the left side of the head; and was so severe as to prevent sleep for many nights, and to deprive her at times of her senses and memory. The blindness of the left eye became complete about the beginning of August: and toward the end of that month the upper eye-lid dropped. This palsy of the levator muscle occurred suddenly; and (as it so often does) in the night-time.

The attacks of headache had been, for the last month, less frequent; but, during that period, she had several times lost all recollection of where she was, and what she was doing. Her deafness, too, had increased: she could not hear the ticking of a watch placed against the left ear; and could only hear it with the right, at a distance of about four inches. The facial nerve was quite unaffected.

January 1st, 1845.—Hardly any change has taken place in the state of the patient since October of last year. Her health is pretty good: but she has occasionally a feeling of general faintness, which seems for a few seconds to pervade her limbs, as if all power in them were gone. Her memory also, now and then, suddenly fails; so that, for a short time, she forgets where she is.

All sight, and even all perception of light, is lost in the left eye; but still no change can be detected in any of its coats or humours, nor are the movements of the eye-ball impaired in any direction except upwards. The paralysis of the *levator palpebræ* continues as before.

March 10th.—I saw the patient again to-day, and was surprised to find her left eye as wide open as the other, and all its movements apparently restored. She told me that, on the morning of the 28th of January (a month after my last visit), her husband noticed that her left eye was partly open; and in the course of the day she could completely lift the lid, which had been quite paralysed the night before. I made a careful examination of the eye, and found that not only the *levator palpebræ*, but the *superior rectus*, also, had regained its power, and that the globe could be turned upwards as perfectly as the right: in short, there was no difference between the motions of the two eyes. The left iris was still in the same condition as on my last visit: the pupil neither dilated nor contracted, and slightly oval in shape. On my drawing up the window-blind, the patient remarked that she could see more light; and afterwards, with the right eye closed, she noticed readily each time that I waved my hand to and fro before the left one. She had, therefore, gained some degree of sensibility of the retina; for the day was dull and cloudy, and, on my formervisit, she could not discern even a ray of sun-light. She told me, also, that "she could now cry with the left eye as well as with the other." When ammonia was snuffed up the left nostril, it took away her breath, and produced a watering of the eye.

Nose.—The left nostril was not so dry as it used to be; but smell was still wanting in it. When

the right nostril was closed, the odour of snuff was not recognized.*

Skin.—The common sensibility of the skin was not in the least restored; the loss of feeling being still rigorously bounded by the median plane; and extending over the left half of the forehead, nose, upper and under lip, and chin; the left eye-ball and lids, and cheek (except the region before alluded to, over the parotid gland and masseter).

Tongue.—For testing the gustatory powers of the tongue, I had provided substances perfectly inodorous, namely:—sugar, common salt, and disulphate of quina; and these, being finely powdered, had the same appearance; so that the patient could not possibly be misled by confounding taste with smell, nor by knowing beforehand what substance was to be tried upon her.

The mouth was first thoroughly rinsed with warm water, and this was repeated after each experiment, until all taste of the substance employed had been got rid of.

Experiment 1st.—I carefully introduced some sugar upon the tip of a wetted finger to the base of the tongue on the left side; at the same time fixing the tongue, so that no other part than that to be tested should come in contact with the sugar. To prevent

* The effect produced by the ammonia is of course to be accounted for by its *punyency* acting upon the membrane of the air-passages—just as it would stimulate the conjunctiva—which is quite distinct from any *odorous property* it may possess.

the necessity of the patient's speaking, and thereby bringing the substances upon the right side of the tongue or upon its tip, I resorted to signals. When I had rubbed the substance on the tongue for a second or two, I asked, "Do you taste anything?" She raised her hand in token of affirmation. "Is it bitter, salt, or sweet?" She moved her hand a second time when I came to the right word. In this first experiment the sugar was tasted at once.

Experiment 2nd.—Salt was as instantly recognized when rubbed on the same part.

Experiment 3rd.—I next rubbed sugar on the left side of the *tip* of the tongue, and about an inch further back—no taste whatever.

Experiment 4th.—Salt on the same part—not tasted.

Experiment 5th.—Sugar on the *base*—right side—tasted instantly.

Experiment 6th.—Disulphate of quina to the front part of the right side—tasted instantly.

Experiment 7th.—Salt to the same spot—tasted.

Experiment 8th.—Quinine to the base: left side—tasted.

Experiment 9th.—(Repetition of Experiment 1st) sugar to the base, left side—tasted.

Experiment 10th.—(Repetition of Experiment 2nd) salt to the base, left side—tasted.

I next tried the tactile sensibility of the tongue. Her eyes being closed, I pricked with a sharp-pointed probe all over the anterior part of the left half of the tongue. She did not know I was touching her: but

as soon as I reached the *base* of the organ on that side, or passed over the median plane, she felt it instantly: sensation being perfect all over the right half of the tongue.

Hearing.—She heard distinctly the ticking of my watch at a distance of four or five inches from the left ear, the right meatus being closed. (Compare the report of February 9th, 1844.)

June 1st.—Since the above observations were made, no change whatever has occurred in the condition of the patient.

A cause might reasonably have been assigned for this affection of the fifth nerve, by supposing a tumour to exist within the cavity of the skull, compressing both the motor and the sensitive portion of the nerve: and the impaired, and occasionally double vision might be accounted for by the indirect pressure exerted on the brain. That the pressure which interfered with the functions of the nerve was applied near its root, or at the Casserian ganglion, was evident, from the fact of the motor portion being equally affected with the sensitive: and that the pressure did not encroach upon the *foramen lacerum orbitale* seemed likely, from there being no enlargement of the branches of the ophthalmic vein, nor any paralysis of the 3rd, 4th, or 6th nerves; parts which could hardly have escaped injury from a tumour pressing upon the cavernous sinus.

When, at a later period, the blindness of the left eye became complete, and the movements of the

globe annihilated, these symptoms seemed easily accounted for, by supposing an increase to have taken place in the bulk of the tumour, whereby the optic nerve and the other parts going into the orbit might have been compressed. The sudden and complete recovery of the motions of the eye-ball and lid, and the partial restoration of vision which took place, were therefore the more unexpected.

In the next case the disease appears to have exclusively confined itself to the fifth nerve, both portions of which were rendered totally unserviceable. There was at no time any impairment of the functions of the motor nerves of the eye-ball; and the loss of sight was due not to disease of the retina, but to the deposit of lymph in the substance of the cornea, in the anterior chamber, and on the capsule of the lens, as the result of inflammation.

The existence of neuralgia in parts quite insensible to outward impressions, (which has frequently been noticed in other cases) illustrates, in an interesting manner, the law by which irritation in the course or at the root of a nerve is manifested at its periphery.

Case 2.—Johanna Harris, aged 59, came to the Ophthalmic Hospital, (October 8th, 1844,) with ulcerations at the inner *canthus* of the left eye, and on the left *ala nasi.* She had followed the very trying employment of shoe-binding since her childhood, and her sight had latterly become so dim that

she could no longer see to work. Her general appearance was that of a person suffering from long-continued pain: her pulse was feeble and slow. The ulcerations had hard, irregular, raised edges, and were remarkably dry, and destitute of secretion on their surface.

On her first visit she was ordered to take the *liquor arsenicalis*; and under this treatment the ulcerations soon healed up.

My attention was now directed to the condition of the fifth nerve, in consequence of the insensibility to pain which the ulcerated surfaces had presented. I found total anæsthesia of the left eye-ball, the Schneiderian membrane of the nostril, the skin of the forehead as high as the vertex, and of the left half of the face, except the ear and the skin over the masseter muscle, where branches of the cervical plexus ramify. The left portion of the tongue supplied by the fifth nerve was also insensible. The patient told me that she had suffered very much about six or seven months ago from tooth-ache, and in consequence had the left canine tooth extracted. About a fortnight afterwards she found on awakening one morning that the left side of the face was numb, and it had continued without feeling ever since.

Eye.—The left eye-ball might be scratched, or the conjunctiva pinched, or touched with nitrate of silver, without the slightest sensation being experienced, and even without producing injection of the vessels of the part.

Tongue.—Finely-pounded sugar, rubbed on the

left side of the tongue *towards its fore part*, was not tasted at all, but *at the base* of the organ on the same side the taste was distinctly perceived. Throughout the right half of the tongue both feeling and taste were perfect.

That the disease had implicated the motor portion of the fifth nerve, was evident from the loose and flaccid condition of the temporal and masseter muscles on the left side, which took no share in the movements of mastication.

All the muscles supplied by the facial nerve were perfect in their action.

Except the healing of the sores on the nose and at the corner of the eye, little change was observed in the patient's condition until

December 20th, when she came to the hospital with the left eye much inflamed. The feeling in the left side of the face was somewhat restored, and the patient could tell, without looking, what part of the skin was touched with a pen. She said that the sense of taste was also returning in the left half of the tongue. The following was the state of the eye. The conjunctiva much and uniformly reddened; the sclerotica also injected, especially around the cornea. The cornea itself was rather dull; towards its lower portion it seemed as if the epithelium had been scraped off: but this appearance was probably the result of a vesication of the corneal epithelium, as there was a transparent *bulla* of the raised membrane still visible upon the upper part of the cornea. The iris was dull, slightly greenish in colour, and

quite uninfluenced by light. The patient felt but very little pain; only now and then, she said, there was a burning sensation in the eye-ball, which soon passed off again. The surface of the eye, which hitherto had been totally without feeling, had had its sensitiveness so much restored, that a slight touch upon the ocular or palpebral conjunctiva was recognised instantly. The sight had become rather worse since the inflammation came on; the hand waved between the eye and the light could only be discerned as a moving shadow.

Cupping to ʒx to the left temple was ordered, and a grain of calomel three times a-day. After the mercury had produced some soreness of the mouth it was discontinued, as no benefit seemed to result from it. The use of quinine was afterwards resumed.

1845. January 14th.—The patient has complained for some days past of occasional sudden and acute pains, which run along the branches of the inferior dental nerve from the mental foramen, along the course of the infra-orbital nerve, and are very severe at the left *ala nasi*, just where the naso-lobal twig comes out from behind the nasal bone.

The livid injection of the conjunctiva around the edge of the cornea continues; the cornea itself is almost bare of epithelium, and looks dry and dull. The iris remains motionless, and of a dirty-green tint; the pupil is contracted, and obscured by lymph. The entire surface of the eye-ball is dryer than that of the right; and, from the commence-

ment of the disease, the secretion of the lacrymal gland seems to have been completely arrested.

21st.—The nervous pains have ceased, and sensation is partly restored in the skin of the upper and lower lip, and in the *ala nasi*, on which parts she could to-day feel the touch of a probe; but the cheek, the eye-lids, and the forehead, are as insensible as ever. The eye is, if possible, dryer than before, and the cornea rather more hazy; but the redness around its border has diminished.

24th.—The injection of the conjunctiva and sclerotica is still less; but the most marked change has taken place in the cornea, which has almost entirely regained its transparency. The eye-ball is still quite insensible. There has been some return of neuralgia since the last report, but only in a slight degree. The treatment has chiefly consisted in active purging, with frictions of mercurial ointment and opium to the left half of the forehead.

February 7th.—The patient did not come to the hospital last week, but returned this morning worse than ever. She is tormented with neuralgia, and the eye is in the following condition: a dull red zone surrounds the edge of the cornea, which is so opaque, especially in the middle, that the pupil cannot be discerned. This opacity is not interstitial, but seems to be caused by lymph upon the posterior face of the cornea. The lower part of the anterior chamber is also full of flocculent lymph, into which red vessels are prolonged.

At this interesting stage of the disease I lost sight

of the patient, and she has not since visited the hospital.*

It would be trespassing too much on the attention of the Society, at the close of so long a paper as the present, if I were to enter into details upon the physiological questions which it suggests. Much has been said and written of late years about the sense of taste, and the nerves by which it is effected. If we find Magendie, Mayo, Müller, Gurlt and Kornfeld, Alcock, Guyot and Cazalis, and Reid, attributing tasting powers both to the glosso-pharyngeal nerve, and to the lingual branch of the fifth; other physiologists, as Valentin, Marshall Hall and Broughton, Panizza and Wagner, are equally positive in restricting those powers to the glosso-pharyngeal alone; whilst Romberg has a theory altogether different from either of the preceding.

The opposite conclusions to which the same kind of experiments, made with great care, have conducted Reid and Panizza, show that much still remains to be done in this branch of physiological

* Since writing the above, I have seen the patient again (August 5th, 1845). All perception of light is lost in the left eye; the pupil being closed by lymph. The cornea, strange to say, has regained its transparency. The eye-ball, like every other part supplied by the fifth nerve, is utterly without feeling. There is palsy of the external rectus and levator palpebræ muscles; and, in a slighter degree, of those supplied by the facial nerve. The patient is quite deaf on the left side. She has frequent attacks of giddiness, and loss of memory. The pain no longer follows the course of the fifth nerve, but is referred to the inside of the skull.

inquiry. I allude to these various theories to account for the balanced opinions with which I made the observations just recorded; not being prepossessed, I believe, by a wish to prove or to disprove any particular hypothesis, but simply desirous of learning as much as I could of a subject both interesting and obscure.

ACCOUNT OF A

CASE OF

EXTERNAL AND INTERNAL CEPHALHÆMATOMA,

COMPLICATED WITH FRACTURE OF THE RIGHT PARIETAL BONE, IN A NEW-BORN INFANT.

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Received March 27th—Read June 24th, 1845.

I HAVE the honour to lay before this Society, the particulars of a case which is interesting, from the comparative rarity of its occurrence, as well as from the illustration it affords of the manner in which nature brings about the cure of similar affections.

On February 8th, 1845, Eliza Wilcox, aged 16 days, was brought to me by her aunt, from whom I learned that the mother of the infant had thrice previously given birth to living children after easy labours, and that Eliza was born after a perfectly natural labour of five hours' duration; that she cried as soon as she was born, sucked well on the following day, and had continued to suck well ever since. It was not till the third day after birth, that a small

swelling, the size of a walnut, was noticed a little to the right of the vertex, extending downwards and forwards over the parietal bone. This tumour had since increased in size every day, notwithstanding which, the child's health was undisturbed, save that she was rather uneasy at night; and, with one exception, her bowels had acted daily since she was born.

The child was well formed, well nourished, perfectly healthy in her appearance, the pupils of her eyes acted readily under the stimulus of light, she cried loudly, and sucked vigorously. Her head was well formed, and neither was the anterior fontanelle nor were the sutures remarkable either for precocious or for tardy ossification. A soft, elastic, non-pulsating, semi-fluctuating tumour, of irregularly oval outline, occupied nearly the whole of the right parietal bone. The greatest prominence of this tumour was just over the parietal protuberance, a little above which, was a slight depression, or furrow; and again, a second prominence, the base of which reached almost to the sagittal suture. The tumour was thus imperfectly divided into two; that nearer the vertex being softer, and more manifestly fluctuating, than the other. A line, circumscribing the base of both tumours, measured twelve inches. They did not anywhere extend beyond the sutures bounding the parietal bone, but the raised edge or ring which ordinarily surrounds the sanguineous tumour of the scalp more or less completely, was nowhere perceptible.

During the examination of its head, the child seemed uneasy, but did not cry, and pressure on the tumour produced no effect on its nervous system.

I directed the application of slips of adhesive plaster round the head, and the employment of a cold lotion.

On February 12th, the child continued well, with the exception of slight diarrhœa, and the tumour had ceased to enlarge since the application of the plaster.

On February 15th the purging had been checked by some powders which I had prescribed. The child seemed well and happy, did not cry or appear fretful. There was no very obvious diminution of the tumours, but that over the parietal protuberance was rather firmer.

On the evening of the same day, the child became very drowsy, had convulsive twitchings of the muscles of the face, and the skin of the surface assumed a yellowish hue. The child sucked heartily, but vomited soon after sucking, and passed an unquiet night. On the afternoon of the 16th, she was taken to church to be christened; while there she yawned deeply two or three times, and when brought home, had an attack of general convulsions, which lasted for about three minutes. During the night she continued very ill, became convulsed at four o'clock in the morning of the 17th, and died in convulsions at six A. M.

At the *post mortem* examination, thirty-three hours after death, I was not permitted to examine any part except the head.

The external appearances were not remarkable. The face was tranquil, the eyes were closed, and there was no discoloration of the scalp.

On dissecting off the scalp from the subjacent cellular tissue, it was found rather closely adherent to it at some points over the right parietal bone, and at those situations presented considerable vascularity, but was elsewhere remarkably bloodless. Successive layers of exceedingly dense cellular tissue, between which yellow serum was infiltrated, were then removed, and the pericranium was exposed. It was of a reddish colour, and very dense texture, but did not present any other remarkable appearance. On opening that tumour which was seated over the parietal protuberance, it was found to be filled with coagulated blood. The red particles had subsided for a depth of about three lines; the dark clot beneath was more than half an inch in thickness, firm in texture, and adhering closely to the bone; especially near that situation where the depression had existed between the two tumours. The other, more distinctly fluctuating tumour, contained semi-coagulated blood, of the appearance and consistence of gooseberry jelly, and somewhat less firmly adherent to the bone than was the clot in the other situation. After this blood had been removed, a semi-circular layer of dense, reddish, fibrinous exudation, about three lines broad, wedge-shaped, with its narrow edge directed inwards, was seen extending along the inner and anterior border of the tumour. It was situated

immediately beneath the pericranium, adhered very closely to the subjacent bone, and formed, so far as it extended, a raised margin around the effused blood. The sensation it conveyed to the finger was very similar to that which is communicated by the osseous ring that usually surrounds these effusions, though its edge wanted something of the sharpness which that presents. The subjacent surface of the parietal bone was rough and uneven, a condition which, moreover, was not confined to the situation but existed over a large extent of the bone. The roughness seemed to be owing to the deposit of new osseous matter, which apparently had formed the medium of the firm connexion that existed between the clot and the surface of the skull.

The frontal and left parietal bone were much more vascular than usual, though the membranous sutures were in a healthy condition.

The sutures were [illegible] parietal bone was [illegible] however, a fissure was [illegible] from the coronal suture [illegible] anterior fontanelle, [illegible] wards. The edges of this fissure [illegible] making the [illegible] stained with [illegible]

On the [illegible] of blood [illegible] more than [illegible]

the whole of the fossa of the parietal bone. The tumour thus formed was of an oval shape, and its surface was slightly irregular. Between the two layers of the dura mater, by which it was covered, were numerous bony deposits, and a ring of newly-formed bone surrounded its base. This ring had not attained the same development in its whole circumference; osseous matter having been abundantly poured out along the frontal margin of the tumour, more sparingly elsewhere, and being entirely absent along its inner border. It appeared to have been formed partly by ossification of the dura mater, partly by heaping up of new bone on the inner surface of the skull. It would have been impossible to ascertain whether the inner table of the skull had contributed, like the outer table, to the process of reparation, by the deposit of new bone, except by the complete removal of the clot, and consequent destruction of the preparation. The clot, therefore, was raised at one edge only, but in that situation the bone was perfectly smooth and unaltered. The blood appeared to have been poured out in successive layers, none of which, however, seemed of very recent date, since all were firmly coagulated, though the red particles had not subsided, but remained equally diffused throughout the clot.*

The rest of the interior of the skull and the dura mater presented no unnatural appearance. There was a depression of the right hemisphere of the

* See Pl. IV. fig. 2.

brain, in a situation corresponding to the tumour. With this exception, the convolutions of the brain presented a perfectly natural appearance, and there was no fluid beneath the arachnoid, nor any injection of its vessels. The whole right hemisphere was pale, bloodless, and very soft; the left was of natural consistence, but its section presented an unusually large number of bloody points. There was no fluid in the ventricles, but little blood in the sinuses, and no other appearance of importance was observed in the brain.

The two features which impart to this case its chief interest are, the fissure of the parietal bone, and the effusion of blood upon the dura mater, both of which have been but rarely noticed. In the absence of any proof of injury having been inflicted on the child, either before, during, or after birth, it is not very easy to account for the fissure of the skull. The fact that the edges of the fissure were quite clean, and presented no signs of any attempt at the formation of callus, may incline some to suppose that it resulted from violence inflicted on the head after birth, and shortly before the child's death. It will be found, however, on reference to other similar cases, that in none were there any indications of a reparative process about the edges of the fissure; a circumstance, by the bye, on which M. Rokitansky insists* as common to fissures of the skull, however produced. The absence, moreover, of all abrasion of the skin, or injury of the scalp, militates against

* Handbuch der Pathol. Anatomie, p. 248.

the supposition that the cranium was fractured after birth, by external violence. It seems most probable that the effusions of blood, and the fissure of the bone, took place about the same time, while the process of ossification going on around the effusion, on the interior of the skull, indicates sufficiently that many days must have elapsed since that occurrence. The annals of legal medicine * contain many instances of injury of the child before birth, and of fracture of its skull by a blow, or some other act of violence inflicted on the mother. In the present case, however, the mother had received no injury for nine weeks before her confinement; and though she was at that time thrown from a cart, yet as she fell on her back, it is unlikely that the fœtus should have suffered from her fall. The probabilities appear to be in favour of the occurrence of the fissure during labour, of which accident there are several cases on record. In many instances of fracture of the skull in natural labour, some contraction of the antero-posterior diameter of the pelvis has existed, while, at the same time, the fœtal skull has been more firmly ossified, and, consequently, less yielding than usual.† In this case, indeed, none of these

* As instances of this occurrence may be mentioned, the cases recorded by Mende, in Henke's Zeitschrift für die Staatsarzneikunde, Bd. iii., s. 277; Adelmann, ibid., Bd. v., s. 295; Becher, ibid., Bd. xxvi., s. 245. Many other cases may be found, by collating the French and German journals.

† Such are the cases related by Jörg, Schriften zur Kentniss des Weibes, Bd. ii., s. 123; Schmidt, quoted by Jörg, lib. cit.,

circumstances existed, but fissures of the skull have been known to take place during easy labours, and wholly independent of contraction of the pelvis, and of any preternatural degree of ossification of the skull.* In such cases, the fractured bone is usually that which was in contact with the sacrum, and the fracture is produced by the uterine action driving the head forcibly against the promontory of that bone. Sometimes, however, the bone in contact with the sacrum escapes uninjured, while a fissure occurs in the presenting bone. To explain this occurrence, it has been suggested by Professor Jörg,† that the head entering the pelvis very obliquely (possibly, as in the case recorded by himself, from too great inclination of the pelvis), is detained against the promontory of the sacrum, while the presenting bone is forced by the violent uterine action so much lower than the other, as to place the membranous connections of the cranium greatly on the stretch, to bend, and finally to break the presenting bone. In the present instance, it is not possible to determine by which of these two processes the injury was inflicted, though probability seems to be in favour of the former.

It may be suggested that the effusion of blood on

s. 130; and E. von Siebold, in his Journal für Geburtshülfe, &c., Bd. xiii., s. 404.

* Such cases are described by V. d'Outrepont, in V. Siebold's Journal, Bd. xiii., s. 400; by Carns, ibid.; by Hoere, ibid., Bd. v., s. 224; and by Danyau, Journal de Chirurgie, Janvier 1843.

† Loc. cit.

the surface of the dura mater was merely the result of hæmorrhage from the vessels wounded by the fracture of the bone. Feist* indeed, and some other writers, have refused to recognise as instances of cephalhæmatoma, those cases in which the effusion of blood was complicated with fracture of the cranial bones. It should, however, be borne in mind, that cephalhæmatomatous swellings, either external or internal, do not by any means invariably co-exist with fissure of the cranial bones; and in the present case, none but a very subordinate share in the production of the effusion can be attributed to the fissure of the parietal bone. Had the extravasation of blood resulted from the escape of that fluid from vessels wounded by the fissure of the bone, the centre of each extravasation might be expected to correspond to the situation of the fissure. This, however, was very far from being the case either with the external or internal effusion, and that tumour which was near to the sagittal suture was quite remote from the fissure, and unconnected with it. Both the fissure and the effusion probably occurred at the same time, and from a similar cause, but the latter cannot be considered as the result of the former. In M. Valleix's valuable work on the diseases of children, is an engraving that illustrates the ease wherewith the blood may be made to transude through the imperfectly ossified fœtal skull. The direction in which the pressure acts during la-

* Die Kopfblutgeschwulst der Neugebornen. Mainz, 1839, 4to, s. 7.

bour, and the greater porousness of the outer, than of the inner surface of the skull, account for the greater frequency of external, than of internal cephalhæmatoma; but the same cause that ordinarily produces the former is quite adequate, if more energetic than usual, to give rise to the latter.

If it be allowable to judge from the number of instances of internal cephalhæmatoma on record, it must be of very rare occurrence. M. Rokitansky indeed, and M. Baron, both speak of it as being by no means unusual. I have seen, however, an account of only eight cases,* besides which I have

* The particulars of these cases are briefly as follows:—

1. V. Siebold's Journal, Bd. ix., s. 43. The patient was delivered by the forceps, but the fœtal head was uninjured. There existed both external and internal cephalhæmatoma. The child died with symptoms of exhaustion on the fifth day.

2. Held, Hecker's Annalen, 1831, Bd. xx., s. 34. Labour natural, except that the funis prolapsed. Child still-born, with sub-aponeurotic and internal cephalhæmatoma. Bone uninjured.

3. Dubois, Article Cephalhæmatoma, in Nouveau Dict. de Med., t. vii., relates a case which occurred to M. Moreau. The skull received no injury, and there was no external cephalhæmatoma. The tumour was situated on the inner surface of the os frontis. Death was preceded by head symptoms.

4. Padieu, in Valleix, Clinique des Enfans Nouveau-nés, p. 541. Child still-born. Fracture of the parietal bone two inches long, external and internal cephalhæmatoma, which communicated readily with each other through the fissure.

5. Hoere, in V. Siebold's Journal, Bd. v., s. 257. Quick labour, fracture of right parietal bone; internal and very slight external cephalhæmatoma. Head symptoms from birth, death on the fourth day.

6. Burchard, de tumore cranii recens-natorum sanguineo. Vra-

found mention of one other case, of which I have been unable to learn the particulars,* and in one instance, in addition to that related in this paper, it came under my notice, associated with slight hæmorrhage into the cavity of the arachnoid.

The case just related, is the only instance with which I am acquainted, in which the process of cure of internal cephalhæmatoma has been witnessed. It presents, however, the closest resemblance to the method which nature follows in the cure of external cephalhæmatoma, which has been seen and described by many observers. An osseous ring surrounded the effusion, the analogue to that which in external cephalhæmatoma encircles the swelling on the outside of the skull, and which conveys to the finger the impression of the bone within its circumference being absent. It is this new formation in an early stage, which has been already described as forming a partial boundary to the upper of the two external tumours. Perhaps the absence of any such ring around the other external effusion may be accounted for by the separation of the dura mater, as well as of the

tislaviæ, 1837, 4to. Case V. is one of fissure of the parietal bone in a premature fœtus, external and internal cephalhæmatoma, death from marasmus in twenty-one days.

7. Case VI. An aperture existed in the bone, of the size of a fourpenny piece; external and internal cephalhæmatoma; death from trismus on the twenty-first day.

8. Case III. An oblong aperture in the bone, external and internal cephalhæmatoma; death in convulsions on the ninth day.

* Betschler, Annalen, Bd. ii., s. 50.

pericranium in that situation, and the consequent impairment of the nutrition of the bone. The bony plates deposited in the dura mater correspond to those which, in other cases, have been found in the pericranium, and at length by their coalescence having completed the transformation of the membrane into bone, have led some observers into the erroneous supposition that the effusion had taken place between the two tables of the skull.

It is fair to infer, from the perfect correspondence of the curative process in this case of internal cephalhæmatoma, with that which is known to take place when the blood is effused on the exterior of the skull, that the remaining steps of the process would have presented the same similarity, if the child's life had been prolonged. The effused blood would doubtless have become absorbed, new bone would have been deposited on the interior of the cranium, adhesion would have taken place between it and the ossified dura mater, and slight thickening of the skull would in process of time have been the only remaining trace of this extensive mischief.

While speaking of the cure of cephalhæmatoma, I may perhaps be pardoned for suggesting a doubt as to whether ossification of the pericranium covering the effusion be, as Prof. Chelius* conceives it to be, of invariable occurrence. I have twice watched the spontaneous cure of cephalhæmatoma most carefully, and found the tumour gradually diminish in

* Medicinische Annalen, Bd. vi., Heft 3; quoted in Canstatt's Jahresbericht für 1842, Bd. i., s. 452.

size, without ever presenting that sensation to the finger, like tinsel crackling under pressure, on which Chelius insists as pathognomonic of ossification of the pericranium. May not this ossification of the pericranium be an exception to the general rule, and the cure be ordinarily effected by the gradual absorption of the blood, and the return of the pericranium to its natural relations, without any alteration of its texture?

The duration of life, and the cause of the death of children who have presented effusions of this kind beneath the cranium, have been very various. Twice the child was still-born; in one instance it died on the fourth, in one on the fifth, in two on the ninth, and in two on the twenty-first day, and in one the date of death is not mentioned. In one of the two cases which have come under my notice, convulsions occurred fifteen hours after birth, and the affection terminated fatally in forty-one hours, a large quantity of blood being found after death beneath each parietal bone. In the other case, which has just been detailed to the Society, no symptom of affection of the brain appeared till the twenty-third day after birth, and death took place on the twenty-fifth.

The yielding structure of the infantile skull, with its membranous fontanelle and unossified sutures, is probably the reason why the effusion of blood upon the surface of the brain does not at first, nor invariably, cause cerebral symptoms. In the present case, the nature of the reparative processes plainly

indicates that the effusion beneath the dura mater must have existed for many days before the symptoms became apparent. It may not be easy to assign a reason for their development at the time when they did eventually come on, but the absence of symptoms in some cases of hæmorrhage into the cavity of the arachnoid in infants, or their tardy supervention long after the blood has been poured out, proves satisfactorily that this feature in the present case, though not explicable, is at least accordant with analogy.

LARGE OPENING

INTO THE

ANTERIOR PART OF THE URETHRA,

CAUSED BY SLOUGHING,

AND ATTENDED BY CONSIDERABLE LOSS OF STRUCTURE, SUCCESSFULLY TREATED BY OPERATION.

WITH REMARKS.

By F. LE GROS CLARK, Esq.,
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Received March 1st—Read June 10th, 1845.

Fistulous openings communicating with the urethra in any part of its course, are alike distressing to the patient and troublesome to the surgeon. Some of these, however, admit of being remedied with comparative facility, either by the employment of caustics or other agents, by which a new surface to the walls of the sinus is procured, and a fresh action established. This observation, however, applies almost exclusively to small apertures, and those which have their seat in the perineal and scrotal portions of the passage, where the integuments and superficial structures are of sufficient density to admit of an active process of granulation, or organization of recent lymph: this appears not to be the case in the thin texture of the urethral walls anterior to the scrotum. It is therefore, in this last-mentioned division of the

urethra, that even small openings have been found to frustrate repeated attempts to close them; and thus, *à fortiori*, the larger deficiencies have been often regarded as incurable. The truth of this remark can scarcely be borne out by stronger evidence than that of Professor Dieffenbach, who, having succeeded in healing an opening in the anterior portion of the urethra, of sufficient size to admit a large catheter, exultingly congratulates himself on hav in arrived at the solution of an enigma which had so long puzzled him, and on having mastered a difficulty which had so often foiled him.

The most efficient method for closing large urethral fistulæ, where it would be vain to hope for success from the simple modelling process, is by a plastic operation,—the integument required for the purpose being obtained from the neighbourhood. Deficiencies in the perineal and scrotal portions of the urethra have been successfully treated in this way: but the same success had not, as far as I can ascertain, attended attempts to close even small apertures in its anterior part, until the ingenious and talented Professor of Berlin, proposed a modification of the usual method adopted, and by which he states he at last succeeded in accomplishing what had so often before baffled him. In his interesting paper upon "Urethral fistulæ,"* Professor Dieffenbach justly attributes the failure of these operations to the

* For convenience of reference, the translation of this paper, in the 10th vol. of the Dublin Journal of Medical Sciences, has been consulted.

presence of the urine, which it is next to impracticable to keep from the edges of a newly-pared wound, communicating with the passage. It appears indeed as if the presence of urine were quite sufficient completely to arrest the adhesive process: and so much importance does the above-mentioned surgeon attach to this obstacle, that he proposes as a question for consideration, whether it might not be deemed admissible to cut into the perineal portion of the urethra for the purpose of passing a catheter, and thus withdrawing the urine by this opening.

But another difficulty, and one which peculiarly applies to the anterior portion of the urethra, is the extreme thinness of its wall and tenuity of the integument in this position: and thus, when the edges are fairly brought into contact, there is not superficies enough to present a prospect of union by the first intention. The above considerations, in the truth of which I entirely coincide, led Professor Dieffenbach to act upon a principle, the justness of which was vindicated by the result. This I think it necessary to refer to, before narrating my own case, as the principle involved is identical, though the mode of proceeding is different.

It has been remarked that the tendency of the *edges* of a wound to unite is proportioned to the extent of surfaces brought into contact; being feeble where the skin is thin, and comparatively vigorous where it is dense and richly supplied with vessels. This axiom being admitted, it is merely a further extension of it to assume that where two

surfaces, instead of edges, can be brought into relation with each other, the adhesive tendency would be proportionately great. With a view of putting in practice this principle, Professor Dieffenbach dissected up the integument on either side of the fistula to some extent, so as to make its pared margins communicate beneath the skin, with lateral incisions, which he made parallel to the urethra; the result of this arrangement being, to allow of the integument contracting fresh surface-adhesions, when shifted inwards on either side to its new position. As the case of the individual on whom I operated presents some points of interest independently of the operation, I shall briefly relate it throughout.

Thomas Hobbs, ætat. 42, was admitted into St. Thomas's Hospital, June 18th, 1844. He is a wine-porter, and has been addicted to free living, as his appearance attests. Sixteen years since, he first contracted gonorrhœa, which he cured by an injection. He several times afterwards had the same disease, and employed a similar measure for its removal; but his last and worst attack was seven years since. He then also had recourse to injection, and stopped the discharge, but produced retention of urine, for which he was admitted into the Westminster Hospital, where bougies were passed, and he was dismissed quite cured in five weeks. He says he then continued well for some time, and contracted no fresh disease; but at the end of two years he began to experience difficulty in

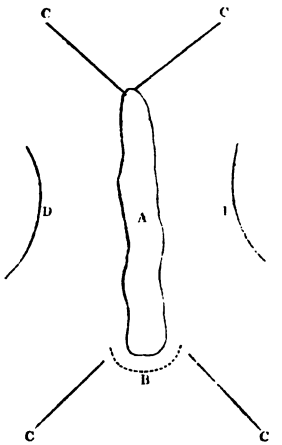
making water; after which the stream gradually diminished, but he sought no advice until the period of his admission into St. Thomas's.

His account of himself then was, that for some time past he had only been able to pass his water *guttatim*, and that he usually occupied about twenty minutes in emptying his bladder; that the urine invariably dribbled away at night; and, further, he affirms, that the semen does not pass forwards along the urethra. Every form, size, and variety of catheter and bougie were tried, but not a single one could be made to pass the stricture, which occupied the membranous part of the urethra, immediately behind the bulb. The only alternative left was an operation, which I accordingly performed on July 15th, by first passing a large catheter down to the seat of stricture, and cutting upon it in the raphe of the perineum: the stricture was subsequently freely divided, and the catheter then passed on into the bladder. Nothing untoward accompanied or immediately followed this operation. The catheter was kept in the bladder, and shortly afterwards replaced by one of elastic gum material. About a week afterwards, he complained of pain in the right iliac region, for which a mustard plaster was applied, and some Dover's and grey powder given: but, on the following day, the pain and tenderness had extended to the cord and testicle, which were swollen and painful. The scrotum was freely leeched, and a calomel purge was ordered. After this he mended, but again fell back, having a renewal of the above

symptoms, but of a much more severe character; the inflammation of the scrotum was very acute, and the constitutional symptoms were alarming, so that it was found necessary to administer stimulants and support, combined with anodynes. He gradually rallied, but not without the separation of a large slough from the junction of the scrotum and penis, by which the urethra was laid open to a considerable extent. The subsequent progress of the patient was very slow; he constantly required his wonted support of wine and brandy, and still the reparative process was inert and tardy. In October, the perineal wound was still partially open: the anterior deficiency of the urethra had cicatrized round and contracted slightly. In November, there being no improvement in the perineal opening, through which very little water passed, as the elastic catheter was still worn, I had caustic applied two or three times, but without much benefit; it was therefore left alone, whilst I addressed myself to the closure of the large anterior opening; anticipating that the cure of the perineal aperture would be more easily dealt with afterwards.

The position of the deficiency (A) which I proposed to close, was in that portion of the urethra anterior to the scrotum, and extending a very little way into the scrotal division; and its length, when the penis was turned up, measured about one inch and a quarter, the two margins being so far separated as to leave the upper wall of the urethra entirely exposed: it presented the bright red, villous

appearance represented in the drawing.* I should add, that each time the patient made water, the urine flowed out freely through this large opening, by the side of the catheter. The following were the steps of the operation.



A large-sized (No. 10) elastic catheter was passed into the bladder, and the stilet being removed, an assistant was directed to hold the penis up against the pubes; with a small scalpel I then first removed an inverted portion of skin (B) which bounded the opening posteriorly at the scrotum.

I next made, in succession, four incisions through the integument, each commencing at a little distance from the extremities of the urethral deficiency

* The paper was illustrated by full-sized, coloured drawings of the parts before and after the operation: the accompanying wood-cut has been substituted, and incorporated with the text. The sketch represents a part of the penis and scrotum, the former being raised to exhibit the position and extent of the fistulous opening, as well as the lines of incision bounding the flaps, A C.

(c c c c): two of these extended, in contrary directions, upwards and outwards on the sides of the penis, the other two downwards and outwards on the scrotum. Each pair of incisions thus included a nearly rectangular piece of skin between them; and a flap on each side was thus marked out, which grew broader as it extended outwards. I then proceeded (*without* paring the edges, on which I did not at all depend for adhesion) to dissect up these flaps, commencing from the margin of the urethra and advancing outwards, so as to raise the two flaps in question. This being effected, I waited for ten minutes or a quarter of an hour, until the surface-bleeding had been arrested, and then undertook the concluding step of the operation, which was to bring these flaps over the urethra, and apply them *surface to surface*, not edge to edge. For this purpose, I had prepared four small quadrilateral pieces of stiff leather, about half an inch square, and perforated. These were applied, two and two, opposite each other, and kept in position by platina wire, which I passed through the leather of each side, and the intervening folds of skin, and fastened by twisting the ends together. In this way the flaps were sustained in relation, forming a ridge which projected a little above the level of the pieces of leather, a small intervening portion being left uncompressed. The operation was completed by a semilunar incision (D) on either side of the penis, parallel to the flaps, in order to obviate the effects of any after tension; and three or four sutures were

employed at the extremities of the wound. The only dressing used was lint dipped in tepid water; and the patient was not allowed the stimulants he was accustomed to, for twenty-four hours.

On the following day, there was some moderate swelling in the borders of the flaps, and some little inflammation in the surrounding parts, sufficient to indicate the value of the lateral semilunar incisions; there was but little uneasiness, and no constitutional disturbance.

On the second day there was a trifling discharge, and the tension of the flaps was relieved by a few punctures which I had made the day previously: the small sutures were removed; no urine had passed by the opening. On the third day, there was a disposition on the part of the lower division of one flap to slough; the lower pieces of leather were accordingly removed. On the fifth day the remaining lateral supports were entirely removed; the partial slough had not extended, and adhesion of the whole length of the flap, with the exception of one small point, was apparently complete. A caustic lotion (argent. nitrat. gr. v. aquæ ʒi.) was applied to the sloughy margin, and in the course of a short time the slough separated. The only point where adhesion had not taken place was at the centre, in the interval between the upper and lower lateral supports; and this was owing to the retraction of that part when the integument began to swell. The lowest extremity of the fissure also remained open, sufficiently to admit a probe. Both of these points

were freely touched once with pure caustic, and soon afterwards closed and healed. Indeed, I question whether the upper point would have required this further treatment, had I been disposed to allow it time to heal by itself. I should have mentioned that the lateral incisions granulated, and soon skinned over.

Whilst this part of the cure was proceeding, I turned my attention to the perineal opening, which still remained in communication with the urethra. I again freely applied caustic, and in a couple of days afterwards, pared the external edges, and kept them in contact by a pin and twisted suture, as for hare lip, but to no purpose. A further and subsequent application of caustic in solution was attended by a sharp attack of inflammation in the left testicle, for which free leeching, and calomel with opium, were necessary: his mouth was affected by the mercury, but the inflammation was subdued. Being disheartened by this result, and fearing to re-produce the mischief I had just succeeded in controlling, by the further employment of caustic, I determined on removing the catheter, and leaving the opening to close, if it would, by itself. The patient passed water freely in a large stream: he felt no pain where the lower wall of the urethra had been renewed, but spontaneously remarked that he had a very curious sensation in the part for the first two or three days, which led him, until undeceived, to believe that the water was running out of the former opening, and over the scrotum. This is no doubt to be explained

by the sensation being referred to the skin which originally covered the penis and scrotum, and which had not yet been identified with the urethra, of which it then formed an integral part: this sensation was not at all painful, and has since gradually subsided.

The earlier history of this case proves that it belonged to that class of slowly-forming and permanent strictures so often referrible to the injudicious use of injections, and which are curable only by cutting or caustic. I gave a preference to the operation of cutting on the stricture from the perineum, on account of its greater certainty, and freedom from dangerous consequences: caustic I have never seen employed. The inflammation which took place about a week after the first operation, and laid the foundation for the second, belonged apparently to that character, which is referrible rather to continuity of texture, than sympathy. The pain and tenderness extended outwards in a retrograde course from the urethra along the vas deferens, until it reached the testicle: and such was likewise the nature of the second attack, which induced me to employ more active means for subduing what previous experience had taught me so much to dread. When the second operation was undertaken, so discouraged was I by the accounts of the failure of similar operations, that I was far from sanguine that I should obtain even a partial benefit from it, and was therefore proportionately gratified at the entire success which attended it.

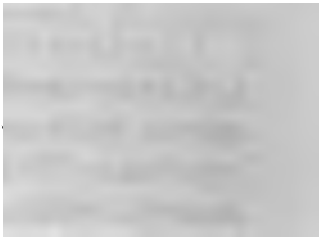
The patient, further, was by no means a favourable subject for a rapid adhesive process: his habits of life, and the artificial stimulus it was necessary to supply him with, to a certain extent proved this. I thought it desirable to use as large a catheter as could be introduced prior to the operation, as the urethra is thus better filled, and there is less chance of the urine finding its way in any quantity by the side of the instrument.

I employed four lateral supports instead of two, hoping thereby to obviate the risk of cutting off the circulation, and strangulating the flaps. I am not sure that I should not prefer a continuous support on either side, if I had another similar case to treat. I did not find that the platina wire employed presented any advantage over a common suture. No urine passed from the wound during the progress of the cure, although it used to pass almost in a stream beside the catheter, prior to the operation. The slough which separated was owing to the compression or strangulation produced by the edge of the leather support, and was very limited. The parts have since been regaining their natural appearance, presenting now merely a slightly raised and irregular ridge along the raphe of the penis: there is no contraction in any direction, and the patient retains his water naturally, and passes it in a full stream, without the slightest uneasiness. The wound in the perineum is now (March 1st) just healed; and I am disposed to attribute this result to the removal of the catheter, rather than to

any other cause: it is possible the mercury which affected the system rather severely for a few days, may have aided in establishing a more healthy action in the part.

Since writing the above, I have observed, for the first time, that Professor Dieffenbach suggests, that the employment of lateral splints might, in some cases, constitute a desirable addition to *his* operation; but he adds, that he has never tried the experiment.

P.S.—I saw Hobbs on June 9th; he then stated that he continued to pass his water in a full and natural stream, and that he had no difficulty in retaining it the usual time. The small perineal aperture, though not quite closed, had contracted, and only allowed of the occasional passage of a drop of urine; this occasioned him no inconvenience; and he treated it as quite unimportant. There is no contraction whatever of the penis, and the cicatrix is scarcely perceptible.



THE

PATHOLOGY OF MENTAL DISEASES.

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In the 26th volume of their Transactions, the Society did me the honour to publish a paper, entitled, "Statistics of Bethlem Hospital, with remarks on Insanity," containing several tables compiled from the Hospital Registers, along with a synopsis of seventy-two dissections of lunatic patients performed by Mr. Lawrence, the surgeon of that establishment. In this communication, I propose to notice one or two points omitted in my former paper, as well as to give an account of thirty-six additional autopsies since made at the same institution.

The first point to which I would advert, is the influence of particular seasons of the year upon mental affections, as illustrated by the following table, indicating the total number of curable insane patients admitted into Bethlem Hospital, discharged cured, and died, during the last twenty-two years, ending the 31st December 1844, arranged according to the quarters.

TABLE OF ADMISSIONS, &c., DURING 22 YEARS.

Season of the year.	Admitted.			Discharged cured.			Died.		
	Males.	Females.	Total.	Males.	Females.	Total.	Males.	Females.	Total.
1st Quar.—Jan. Feb. March	451	649	1,100	139	257	396	39	35	74
2nd Quar.—April, May, June	545	842	1,387	215	375	590	21	34	55
3rd Quar.—July, Aug. Sept.	551	798	1,349	265	410	675	32	30	62
4th Quar.—Oct. Nov. Dec.	471	668	1,139	333	561	894	32	32	64
Totals.	2,018	2,957	4,975	952	1,603	2,555	124	131	255

According to the above statement, a much larger number of insane patients were received during the second and third quarters, than at any other period of the year; that is, when the temperature of the weather increased, so did mental diseases become more frequent. For example, during the second and third quarters of the period referred to, the total curable lunatics admitted into Bethlem Hospital amounted to 2,736; whereas, during the first and fourth quarters, that is, in the cold season, the actual number was only 2,239; being a diminution of 497 patients, or 22 per cent. less upon the whole admissions. In regard to particular months, it may be mentioned that the greatest number of curable lunatics admitted, took place during May,—the fewest in January.

Respecting the curability of insanity, it also appears from the above table that fewer insane patients were discharged cured during the early part of the year, than in the autumnal months, or towards the approach of winter. For instance, in the first and second quarters, the number of cures were under the average proportion, only 986 lunatics having been discharged convalescent during that period; whereas, during the last two quarters, 1,569 patients left the institution free from mental disease, thus making 583 more recoveries, or an increase of 57 per cent. on the total number of cures reported during the latter, than in the first six months of the twenty-two years comprehended in the previous table.

With reference to the mortality of mental diseases, the same document shows that not only the relative proportion of deaths to the total admissions was larger, during the first than in any subsequent quarter, but the actual number of cases terminating fatally during the former period, exceeded the ratio of any other three months of the same series, whilst the fewest deaths occurred during the months of April, May, and June; when, as already stated, more insane patients were admitted into Bethlem Hospital than at any other season.

Supported by these data, the physician may rationally conclude that as the temperature of the weather diminishes, and the year draws to a close, so may he give a more favourable opinion respecting the progress of cases of insanity. At the same time, seeing mental affections are in a greater degree

prevalent during summer than in winter, every exciting cause, whether physical or moral, ought to be then carefully guarded against, particularly in persons who have been previously afflicted with mental alienation.

As an illustration of the improved system at present followed in all well-regulated lunatic asylums, of affording occupation and amusement to the inmates, it may be mentioned that of 422 insane patients under treatment in Bethlem Hospital, during the present or third week of this current month of June, 288 were engaged in some kind of employment, thus making upwards of 68 per cent. who were beneficially occupied, instead of passing their life, as formerly, in listless inactivity.

In connection with this subject, it may be stated, that the more such measures are promoted, the less need, in all probability, will there be for employing personal coercion; and in confirmation of this opinion, I refer to the following table, showing the average weekly number of patients under restraint during the last five years, premising that the system of employing the lunatic inmates of Bethlem Hospital has been more fully developed in each succeeding year.

RETURN OF PATIENTS UNDER RESTRAINT.

Year.	Average Weekly No. of Patients under Restraint.
1840	13½
1841	9
1842	3
1843	3
1844	1½

Considering the number of violent, dangerous, and suicidal patients annually admitted, the facts now stated demonstrate, in a very decided manner, the advantages of the new system, and fully confirm the opinions entertained by those who advocate the non-restraint mode of treating the insane.

Desirous of contributing some facts towards elucidating the pathology of mental diseases, I now beg to bring before the Society the subjoined report of thirty-six dissections of insane patients, recently performed at Bethlem Hospital, thus making 108 autopsies, including the seventy-two contained in my previous paper, which I hope will be thought worthy of perusal by the Fellows, and those members of the profession who take an interest in similar investigations.

SYNOPSIS OF DISSECTIONS.*

No. 1.—Female, æt. 31. In hospital eight months.

Head.—Skull-cap thick, and unusually heavy. Considerable and general infiltration of pia mater; also of sub-arachnoid, at base of brain. Around optic nerves, crura cerebri, pons Varolii, and commencement of medulla, fluid had the yellow colour and consistence of thin pus, being also fœtid. A considerable quantity of this puriform offensive fluid

* When the chest or abdomen are not mentioned, it should be understood, that no diseased alterations of structure were observed in either of these cavities.

remained in base of skull, after brain was removed. Infiltration had a similar puriform character, in a few spots, over cerebral hemispheres, particularly on their exposed flat surfaces; substance of brain soft in these situations. Lateral ventricles enlarged, and distended with a slightly opaque fluid.

Abdomen.—An abscess on convexity of right ilium, containing offensive pus, with a large cellular slough. Peritoneum, both in its parietal and visceral portions, thickly covered with minute tubercles. Liver universally and closely adherent to diaphragm.

No. 2.—Female, ætat. 42. In hospital three months.

Head.—The large head of this patient was of a deep livid colour, from the highest state of vascular congestion. All the external vessels extremely turgid, and much blood flowed on dividing the scalp. Blood-vessels of bone and dura mater in same state, and those of former structure exhibited an unusual degree of injection. Very slight infiltration of pia mater, on convexities of cerebral hemispheres, and a slight increase in the quantity of ventricular fluid.

Chest.—Lungs did not collapse when thorax was opened. Heart large, particularly left ventricle, which was also thick and firm.

No. 3.—Female, ætat. 43. In hospital three weeks.

Head.—External vessels of head, bone, and dura mater, empty. Veins of pia mater turgid. Cellular texture of that membrane greatly infiltrated.

Several convolutions on convexities of cerebral hemispheres considerably shrunk, the spaces thus left being occupied by infiltrated pia mater. Lateral ventricles distended with limpid fluid, much fluid about velum, both in its upper and under surface, and in base of skull, after brain was removed.

No. 4.—Female, ætat. 23. In hospital eleven days.

Head.—Blood-vessels of brain moderately turgid; serous infiltration of pia mater, covering cerebral hemispheres, and more than an ounce of perfectly transparent fluid in lateral ventricles, and in base of skull, after brain was removed.

Chest.—Greater part of right lung congested, and œdematous. On posterior lobe, about an inch and a half had mortified; it was perfectly flabby, and yielded on incision, a dark putrid sanies.

No. 5.—Male, ætat. 43. In hospital ten months.

Head.—Vessels of brain and membranes loaded with blood; great serous infiltration of pia mater, and much fluid remaining in base of skull after brain was removed; lateral ventricles enlarged and distended with about three ounces of clear fluid.

Chest.—Left lung universally connected to thorax by old adhesions.

Abdomen.—Extensive inflammation of peritoneum, both in parietal and visceral portions; ilium, colon, and omentum, adherent; pelvis contained four or five ounces of an offensive dark-coloured fluid, partly purulent. On concave surface of spleen, a crack, about an inch in length, through fibrous and serous coats, the organ being rather

large, and of a deep leaden hue. Three or four ounces of dark offensive fluid in left hypochondrium.

No. 6.—Male, ætat. 42. In hospital four days.

Head.—All the vessels of the skull, membranes and brain loaded with blood to the utmost degree; those of the bone filled to minutest ramifications, so as to give the bony substance a general, and rather deep livid tinge. Detachment of skull-cap required great force, adhesions of dura mater being unusually firm. On separation, black blood flowed from vessels of membrane abundantly at all points. Moderate serous infiltration of pia mater, covering cerebral hemispheres.

No. 7.—Female, ætat. 49. In hospital seven years.

Head.—Vessels of brain and membranes turgid; considerable serous infiltration of pia mater, and much fluid in ventricles.

Chest.—Both lungs extensively tuberculated with numerous small vomicæ.

No. 8.—Male, ætat. 50. In hospital six months and a half.

Head.—Skull-cap heavy; bone, where thickest and diploë most abundant, had a livid colour from turgidity of blood-vessels. Mr. Lawrence observes, "This heaviness and vascular condition of bone are frequently noticeable, particularly in chronic cases." Dura mater adherent to bone so firmly, that it was difficult to detach skull-cap, and membrane considerably torn on separation; arachnoid covering hemispheres partially opaque; considerable serous in-

filtration of pia mater, both on surface of hemispheres, and in intervals of convolutions, which had undergone partial shrinking; lateral ventricles enlarged and filled with pellucid fluid, about two ounces in each cavity, and much fluid remained in base of skull, after brain was removed.

Chest.—Slight effusion of fibrine on right lung, with adhesions to parietes of thorax; small firm bodies of irregular figure, scattered in considerable number over surface of lung and pleura costalis on both sides; they were smooth, of an irregular or round form, not exceeding a quarter of an inch in length, and varying from that, to the size of a mustard seed, semi-transparent, and approaching to cartilage in texture and consistence.

Abdomen.—Great omentum had undergone a change of structure, which Mr. Lawrence remarks, "he had not seen previously." It was thickened, soft, and tolerably smooth, but raised over the whole surface into irregular prominences of various size. This change was caused by the deposition in its texture of a gelatinous substance, yellowish, transparent, and closely resembling calves-foot jelly. Loose edge of omentum irregularly fringed by this effusion, which also raised its general surface into the prominences already noticed, appearing, on first view, as a gelatinous matter lying on omentum.

No. 9.—Male, ætat. 42. In hospital four months and a half.

Head.—Much blood escaped in dividing soft parts, and sawing through cranium. All the blood-

vessels of bone, membranes and brain extremely turgid. Dura mater covering left hemisphere, partially lined by an adventitious membrane, which covered left side of falx nearly throughout, and was continued for two or three inches on neighbouring portion of membrane; its internal surface was smooth, like serous membrane, and inadherent; external part adhered uniformly to dura mater, but could be easily peeled off, leaving that membrane apparently smooth and entire. New production as thick as dura mater, or indeed rather thicker in middle portion, but becoming gradually thinner at circumference, until lost on surface of that membrane; it was rather close in texture, compact, and firm enough to appear a regular membranous production, but could be torn without much force; it presented no appearance of fibrous arrangement, was of a dull brownish red, arising from numerous blood-vessels, of which the larger could be seen with the naked eye. Arachnoid covering cerebral hemispheres, thickened and opaque; cellular texture of pia mater infiltrated. Each lateral ventricle contained about two ounces of clear fluid; septum lucidum rendered thin by distension, and it had given way at the back part, so as to be reduced to a few shreds, between which the two ventricles communicated freely; fornix raised in front, so that the ventricles communicated directly by foramen of Monro. Substance of brain firm, although the examination was not made until third day after death.

Chest.—Posterior part of left lung congested to a considerable extent, and only partially permeated by air. Pleura covering condensed portion of lung slightly inflamed, and about six or eight ounces of rather turbid fluid in cavity.

No. 10.—Female ætat. 29. In hospital three weeks.

Head.—External vessels of head quite empty. All the internal vessels extremely turgid, those of cerebral substance minutely injected throughout; very slight infiltration of pia mater on convexities of cerebral hemispheres. About an ounce of clear fluid in each lateral ventricle, much fluid about velum and pineal gland.

Chest.—Right cavity contained nearly a pint of yellow sero-purulent fluid. Both portions of pleura universally inflamed, and partially covered by soft, yellow fibrine. Inferior lobe of right lung in a state of incipient hepatization.

Abdomen.—Ilium and cæcum of a dark livid red, with superficial ulcerations on colon.

No. 11.—Female, ætat. 40. In hospital three months and ten days.

Head.—Opacity of arachnoid, covering convexities of cerebral hemispheres. Considerable serous infiltration of pia mater; increased quantity of fluid in lateral ventricles. Blood-vessels of brain and membranes turgid; those of cerebral substance considerably injected. Irregular patches of livid red discoloration, so faint as to be just discernible, observed on cut surfaces of cerebral medullary matter.

Chest.—Lungs universally connected to thorax by old adhesions.

No. 12.—Male, ætat. 37. In hospital three years and a half.

Head.—Slight opacity of arachnoid, and partial infiltration of pia mater. Minute injection of vessels of brain. Increased quantity of fluid in ventricles.

No. 13.—Male, ætat. 33. In hospital twelve days.

Head.—Vessels of head, both internal and external, loaded with blood, and those of brain minutely injected; partial thickening and opacity of arachnoid, with infiltration of pia mater; increased quantity of fluid in ventricles. Much fluid remained in base of skull after brain was removed.

Chest.—Sternum broken across, and cartilages of third pair of ribs nearly detached from the breast bone, which had occurred previous to admission. Extensive suppuration round fractured sternum, both before and behind, with a large quantity of thick greyish matter between integuments and bone. The inflammation extended behind cellular texture of anterior mediastinum, which was infiltrated with pus. Similar changes of structure in cellular texture on surface of pericardium. Left pleura violently inflamed, and extensively covered by recently effused soft yellow fibrine. Sixth true rib, on left side, fractured, and matter effused round broken ends of bone.

No. 14.—Female, ætat. 42. In hospital eight years and nine months.

Head.—A considerable quantity of blood, nearly unmixed, had run out of mouth into the shell in which the body was placed. Much blood also escaped in making incisions for opening skull. All the vessels, both of membranes and brain, loaded with blood.

Chest.—Air-cells of inferior lobe of right lung filled with blood, nearly throughout. Partial old adhesions between pericardium and left side of heart; the mitral valve thickened and indurated, and forming a ring of nearly cartilaginous consistence. Fibrine of a dirty grey colour and soft consistence, effused in left auricle. Small deposition between fasciculi of muscular fibres. A similar deposition, but in smaller quantity, in corresponding part of right auricle.

Abdomen.—Liver small, and presented throughout the appearance called nutmeg-liver.

No. 15.—Female, ætat. 45. In hospital six weeks.

Head.—Not a drop of blood escaped in exposing and sawing through skull-cap. Vessels of brain and membranes extremely turgid. Mr. Lawrence states, "he never saw the bloody points, on cutting into cerebral substance, more numerous or larger than in this case." Medullary matter had a faint pink tint, nearly throughout. Slight thickening of arachnoid, in convexities of hemispheres. Infiltration of pia mater. Slight increase of fluid in ventricles,

and much fluid remained in base of skull, after brain was removed.

Chest.—Right lung connected to thorax, by close old adhesions.

Abdomen.—Spleen adhered to diaphragm; its fibrous coat thickened and opaque.

No. 16.—Male, ætat. 33. In hospital fourteen months.

Head.—Blood-vessels of brain somewhat distended. Serous infiltration of pia mater. Increased quantity of fluid in ventricles, and fluid in base of skull, after brain was removed.

Chest.—Both lungs extensively tuberculated, with several vomicæ.

No. 17.—Male, ætat. 48. In hospital eight weeks.

Head.—All the vessels of brain and membranes very turgid. Serous infiltration of pia mater. Increased quantity of fluid in ventricles, and in base of skull, after brain was removed.

Chest.—Both lungs œdematous, and connected to thorax by firm old adhesions.

No. 18.—Female, ætat. 41. In hospital one year.

Head.—Vessels of brain and membranes very turgid. Slight infiltration of pia mater.

Chest.—Upper and back part of left lung firmly adherent to thorax, and tuberculated in five-sixths of its extent, with several vomicæ.

No. 19.—Female, ætat. 39. In hospital eleven weeks.

Head.—Blood-vessels of brain and membranes

very turgid. Bloody points numerous and large on cutting substance of brain, which was partially mottled with a bluish rosy tint. Pia mater infiltrated. Lateral ventricles distended with clear serum, and much fluid in base of skull, after brain was removed.

Chest.—Old adhesions of lungs. Two bony deposits in right lung, one as large as a marble.

No. 20.—Male, ætat. 29. In hospital two years and a half.

Head.—All the vessels of brain and membranes turgid; those in cerebral substance large and full of blood. Arachnoid thickened and partially opaque, over whole of cerebral hemispheres. General serous infiltration of pia mater. Ventricles large, and distended with clear fluid. Much fluid remained in base of skull, after brain was removed.

Chest.—Right lung strongly adherent to thorax throughout, and consolidated by tubercular depositions.

No. 21.—Female, ætat. 26. In hospital eleven days.

Head.—Very slight serous infiltration of pia mater. Vessels of cerebral substance rather larger than in normal state, showing previous increased activity of circulation.

No. 22.—Male, ætat. 59. In hospital eight years and ten months.

Head.—Opacity of arachnoid on convexities of cerebral hemispheres. Infiltration of pia mater. Distension of cerebral vessels.

inflammation of peri-

In hospital two

and those of body
Vessels of brain tur-
inflammation of pia mater.
with effusion of
sides. Three or four
in each pleura. Nu-
excavations. Gan-

In hospital seven

and heavy. Brain, par-
upper part, remarkably
some quantity, effused
covering to dura mater at
without connection to pia
quantity did not exceed a
space of about an inch
about five or six times as
middle fossa of basis
backwards and for-
frontal bones. It was a
and adhered to internal
mater, when cleaned by
covered. Neither breach
could be detected, and
external soft parts,
Considerable serous

infiltration of pia mater. Lateral ventricles contained about twice their normal quantity of fluid.

Chest.—Left lung connected to thorax by old and firm adhesions. Pericardium considerably inflamed, and contained nearly an ounce of light yellowish turbid fluid.

Abdomen.—Membranous coverings of liver partially thickened, opaque, and connected to diaphragm by very strong adhesions.

No. 25.—Male, ætat. 52. In hospital six years and eleven months.

Head.—All the blood-vessels of head, bone, membranes, and brain, in highest state of turgescence, being filled with blood to minutest ramifications. Bloody points numerous on cut surfaces of cerebral substance. Slight serous infiltration of pia mater.

Chest.—Lungs connected to thorax by extensive adhesions. Right lung consolidated, and excavated by a large irregular cavern.

Abdomen.—Liver enlarged, granulated, firm, and greasy in texture, having the appearance observed in dram-drinkers.

No. 26.—Female, ætat. 41. In hospital sixteen months.

Head.—Considerable serous infiltration of pia mater on cerebral hemispheres. Convolutions partially shrunk. General fullness of vessels of brain, and numerous bloody points on all its cut surfaces.

Chest.—Extensive tubercular disease of both lungs, with adhesions on right side, apparently of recent formation.

Abdomen.—Cavity contained about two pints of a turbid fluid, mixed with a few thin flakes of fibrine.

No. 27.—Female, ætat. 38. In hospital seventeen days.

Head.—Vessels of brain rather turgid. Ventricles greatly enlarged, and filled with clear, colourless fluid, particularly in the two lateral cavities, each of which contained about three or four ounces. Anterior part of fornix raised so as to convert foramen of Monro into a large direct communication between the two ventricles. Septum lucidum so stretched by distension of ventricles as to be greatly increased in depth, and converted by removal of its medullary portion into a thin mealy partition, traversed by blood-vessels. Third ventricle distended. Optic thalami universally separated and soft, commissure proportionally elongated.

Chest.—Left cavity of pleura contained a few ounces of puriform fluid, and membrane inflamed. Old adhesions of lungs, posterior lobes consolidated, and exhibited deposits of thick yellow matter.

Abdomen.—Old adhesions of liver to diaphragm, ovaries adherent to uterus and broad ligaments. Peritoneum thickened.

No. 28.—Female, ætat. 46. In hospital four days.

Head.—Frontal bone in its middle part was about twice the regular thickness, presenting a very irregular surface raised into prominences, the size of a large pea; fissured and excavated at one part, so as to make the bone preternaturally thin. Dura mater adhered so firmly, that it could not be stripped off

in the usual way. Surface of brain and the immediately investing membranes opposite to this portion appeared natural, but the blood-vessels of membranes and brain, both on surface and throughout substance of organ, were turgid. Arachnoid partially opaque on convexities of cerebral hemispheres, pia mater infiltrated. Ventricles enlarged and distended with at least three ounces of clear fluid in each lateral cavity.

Chest.—Lower part of left lung in a state of recent gangrene. Other portions of left lung, and also of right, similarly affected, but not to the same extent. Cavity of pleura contained about three ounces of darkish fœtid fluid. Old adhesions on both sides of thorax.

No. 29.—Male, ætat. 65. In hospital eight months.

Head.—Bones of skull thick and heavy. Sagittal suture and diploë in many parts obliterated. Frontal bone dense, and presented irregular projections over the anterior cerebral lobes. Dura mater under thickened frontal bone on left side thinner than natural, and transparent. Arachnoid somewhat opaque. Considerable effusion in pia mater; cerebral convolutions firm and compressed. Ventricles contained some clear fluid. Foramen of Monro large.

Chest.—Old adhesions with small gritty particles on surface of lungs. Pericardium adhered firmly to heart, which was larger and softer than natural.

No. 30.—Male, ætat. 41. In hospital eleven months and a half.

Head.—External vessels of head, cranium, and dura mater, empty; vessels of brain full of blood, and

bloody points numerous on cut cerebral surfaces. Arachnoid of cerebral hemispheres partially opaque. Pia mater considerably infiltrated. Increased quantity of fluid in ventricles.

Chest.—Both lungs tuberculated and excavated by caverns. Recent adhesions of left lung, and old adhesions of right.

No. 31.—Male, ætat. 45. In hospital six days.

Head.—Bones of skull vascular, and dura mater adhered firmly. Arachnoid opaque upon tentorium and under posterior lobe of right side, the two surfaces being adherent. Pia mater infiltrated. Cerebral hemispheres somewhat compressed, and substance turgid. Cortical part congested and pink-coloured. Numerous bloody points on cut medullary texture. Ventricles enlarged, containing about two ounces of clear fluid. Foramen of Monro large. Thalamus opticus, corpus striatum, and cerebellum, turgid, and of a pink colour. Brain generally firm.

Chest.—Lungs emphysematous, dark coloured, and so soft as easily to break down when handled. Pleura adhered extensively to back part of thorax by old adhesions.

No. 32.—Male, ætat. 44. In hospital six days.

Head.—Skull extremely thick and heavy, with rounded projections on inner surface. Sutures obliterated, and dura mater firmly attached. Arachnoid thickened and opaque, with considerable infiltration of sub-arachnoid spaces. About ʒiii of clear fluid in ventricles. Brain generally firm.

Chest.—Some fluid in cavities of pleuræ. Firm old adhesions on right side, converted into a layer of rough cartilage, about the size of palm of hand.

No. 33.—Male, ætat. 23. In hospital five years and three months.

Head.—Skull thin, and not much blood in its vessels, or dura mater. Brain turgid, and cranial contents generally compressed. Surface of arachnoid dry, thickened, and slightly opaque nearly throughout. Slight serous infiltration of sub-arachnoid tissue on hemispheres. Yellow discolorations in six or eight parts on surface of cerebral hemispheres, produced by interstitial deposition into pia mater, which adhered so firmly to cerebral substance, that it was lacerated when detaching them. Change of structure appeared to greatest extent in pia mater covering corpus callosum, which was converted into a thick, tough, and yellowish mass, adhering most firmly to inferior edges of hemispheres, and to corpus callosum. Cerebral substance near this mass was reddened, and filled with small portions of coagulated blood, so as to present an ecchymosed appearance, to a quarter of an inch in depth. Lateral ventricles contained about ʒiii of slightly turbid fluid. Roof of these cavities, corpus callosum, septum lucidum, and fornix, softened, almost to the consistence of thick cream, but of their natural white colour. Rest of brain of normal firmness. Arachnoid about infundibulum, thickened and confused with pia mater, which adhered closely to adjacent parts.

Chest.—Extensive tubercular disease of both lungs, which were excavated by caverns. Upper lobe of left lung in the congestive stage of inflammation.

No. 34.—Male, ætat. 48. In hospital seven months and seventeen days.

Head.—Much blood escaped in dividing external parts, and sawing bone. Vessels of brain turgid. Arachnoid partially opaque, on cerebral hemispheres. Considerable serous infiltration of pia mater.

Chest.—Recent inflammation of right pleura, and about ʒvi of brownish opaque fluid effused, with a small quantity of soft yellow fibrine on surface of pleura costalis, at two points. Left lung congested and inflamed. Firm old adhesions of pericardium to heart.

Abdomen.—Descending colon, instead of forming sigmoid flexure, crossed on front of spine, above brim of pelvis, over right sacro-iliac symphysis, and then descended on right side of sacrum; the portion of intestine, crossing abdomen, being connected to spine by old adhesions.

No. 35.—Male, ætat. 57. In hospital two months and eighteen days.

Head.—Skull-cap thin, and dura mater adhered to it with unusual firmness. Some serous infiltration of pia mater. About ʒij of clear fluid in lateral ventricles.

Chest.—A thin, narrow shred of fibrine effused on surface of right lung, which was loaded with

blood, dark coloured, and preternaturally firm; the centre sunk in water. Coats of coronary arteries partially thickened.

No. 36.—Male, ætat. 41. In hospital three weeks.

Head.—A considerable quantity of blood escaped in exposing and dividing cranium. Skull-cap thick and heavy. Blood-vessels of bone, membranes and brain turgid. Arachnoid, over whole of cerebral hemispheres, and at base of brain, thickened, hardened, and partially opaque, so as to give the surface a slightly milky appearance. On stripping off arachnoid and pia mater, the united membranes were thicker and much firmer than usual. Considerable serous infiltration of sub-arachnoid tissue on hemispheres.

Chest.—Lungs did not collapse when thorax was opened.

According to the above statement, some diseased alterations of structure, more or less evident, in the brain and membranes, were observed in all the thirty-six dissections now detailed; of which the following summary may be given: In thirty-three cases the pia mater was infiltrated. In thirty, there was turgidity of the blood-vessels of the brain and its membranes. In twenty-six, effusion of water had taken place in the ventricles. In sixteen, there was thickening and opacity of the arachnoid coat. In twelve, fluid was met with at the

base of the brain. In nine, the consistence of the brain was altered from its normal condition. In eight, patches, or bloody points, appeared on the cut medullary surfaces. In five, the colour of the medullary, or cortical substance, was altered from its healthy hue to a pink, mottled, or rosy tint; and in four cases, blood was effused in the brain, besides other morbid changes of structure of a less important character; for an account of which I would refer to the synopsis, to avoid superfluous repetition. The same may be said in regard to the cases exhibiting diseased alterations of structure in the organs of the chest; of which description there were thirty examples; being five-sixths of the dissections contained in the present series; whilst only twelve patients, or one-third, showed any morbid appearances in the abdominal viscera.

I subjoin a short abridgment of the pathological changes met with in the brain and membranes of the one hundred and eight autopsies reported in the present and previous communication to the Society. I may remark, that infiltration of the pia mater was observed in ninety-two cases. Turgidity of the blood-vessels existed in eighty-nine; fluid was effused in the ventricles in sixty-seven; effusion had taken place at the base of the brain in thirty-nine. There was thickening and opacity of the arachnoid coat in thirty-two. Bloody points were observed on the cut surfaces of the medullary substance in twenty-seven. The colour of the brain appeared changed in nineteen; and in seventeen cases, blood

was effused within the cranium. These data indicate unequivocally, that the morbid alterations of structure, characteristic of insanity, which pathologists may expect to find in a majority of cases, will be, infiltration of the pia mater, turgidity of the blood-vessels, and effusion of fluid in the ventricles.

Unwilling to occupy the attention of the Society with further remarks, I would in conclusion observe, that although the mental maladies of insane patients demand special attention, their bodily complaints frequently become of such importance as to endanger life, when they must be treated according to general principles. On the other hand, the physician, however conversant with the pathology and treatment of physical diseases, ought also to study the nature and management of maladies of the mind, in order to be prepared for any contingency which may occur in the practice of his profession.

ON SOME OF THE CAUSES OF PERICARDITIS, ESPECIALLY ACUTE RHEUMATISM, AND BRIGHT'S DISEASE OF THE KIDNEYS;

WITH INCIDENTAL OBSERVATIONS ON THE FREQUENCY AND ON SOME OF THE CAUSES OF VARIOUS OTHER INTERNAL INFLAMMATIONS.

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In inquiring into the causes of pericarditis, as well as into many other particulars in its history, it will be found practically important to divide the cases into the two following classes:—

1st. Those occurring in persons previously in good health, or in the course of some sthenic acute disease, as acute rheumatism: and,

2nd. Those occurring in persons previously in bad health, or in the progress of some chronic disease.

The examples of pericarditis which I shall examine more particularly, are twenty-five in number;

and of these, *fifteen* belong to the first, and *ten* to the second, of the two classes just described.

1. *Of the observed causes of pericarditis.*

Thirteen out of the *fifteen* cases, in the first class, occurred in the progress of acute rheumatism; in a fourteenth case, *slight swelling* and *redness* of one or two of the joints of the fingers appeared for a *short* time only, and several days after the pericarditis (which was both *severe* and *fatal*) began. In the remaining case, there was no *obvious rheumatism*; but the patient had been twice the subject of acute rheumatism, some years previously; no exciting cause for the present illness was assigned, and he had profuse perspirations (whether of a sour odour or not, is not stated); whence we may, I think, infer the *possibility* (which facts subsequently to be noticed will perhaps raise to a *probability*) that this was an example of *rheumatic fever without arthritis.* (Vide p. 519). In the fourteenth case we have seen an example of severe and fatal pericarditis, the rheumatic character of which was shown only by the most *trifling* and *transient articular* affection, which might have been easily overlooked. Why, therefore, may the last case not be an example of the same kind, in which the articular affection was either *altogether absent*, or too insignificant to attract attention?

Among these cases, all of which, with the above explanation, were complicated with rheumatism, there were none in which any other cause for the pericarditis could be assigned. One of these conse-

quences, therefore, seems necessarily to follow, viz., either the internal inflammation must be ascribed to the rheumatism, or it must be ascribed to an unknown cause. In the latter case, the rheumatism and the pericarditis may have been wholly unconnected; or they may have been *distinct* effects of this *unknown* but *common* cause.

Out of the *ten* cases contained in the *second* class, *six* had Bright's disease of the kidneys, and a *seventh* had *probably* the same disease, for he had been losing flesh during several months previously; he drank both ale and spirits habitually, and to excess; and just before the attack of pericarditis he was intoxicated, and in that condition got wet, and kept on his wet dress. The urine was not tested, but the kidneys were evidently *diseased*, being increased considerably in volume and weight (nearly 7 oz. each); the distinction between the medullary and cortical portions was less obvious than usual, and the left kidney was lobulated.

In *three* there are no facts enabling me to determine what was the condition of the kidneys. One of these patients had acute rheumatism, and this is the only example among the ten cases of this class in which acute rheumatism existed together with, or near the time of, the pericarditis: it is likewise the only one of the cases in which any other cause than the disease of the kidneys could be assigned for the pericarditis.

There was no evidence of the existence of renal disease in any of the fifteen cases of the *first class*.

In *seven* of them, the *absence* of any such disease was *ascertained*; in six others, the *probable* absence can be shown; and in the remaining two, there is nothing stated that can lead to a suspicion of the kidneys being affected.

In respect to the causes of pericarditis then, a most important difference is observable between the cases which have been g d in the first, and those in the second class. -thirds of the latter were *known* to be complicated ith Bright's disease of the kidneys, and *all* of them *may* have been; but *one case* only was complicated with acute rheumatism, whereas cases n the former class were compli ac r umatism, and none of them, s with renal disease. These facts, which I terwards confirm by others, seem to require us to recognize precisely the same relation between Bright's disease of the kidneys and pericarditis in the one class of cases, which we have already recognised as existing between acute rheumatism and pericarditis in the other class.

Besides the cases of pericarditis noticed above, I have notes of a few others not included in this analysis—e. g. 1. Of several slighter and of two severe cases, all occurring in the course of rheumatism. 2. Of two other severe ones unconnected with rheumatism, but complicated with Bright's disease. 3. Of *one* case complicated with malformation of the heart, and consequent cyanosis. This patient died at the age of eight months, and there was neither

rheumatism nor disease of the kidneys. 4. In the year 1841 I witnessed, accidentally, the inspection of the body of a female who died in University College Hospital whilst under the care of my colleague, Dr. Williams. In this case there was found abscess of the left lobe of the liver, with adhesion of the liver to the diaphragm, pericarditis, pleurisy and pneumonia. There had been no rheumatism, and there was no evidence of disease of the kidneys. The urine had not been examined. The inflammations within the chest were considered by Dr. Williams, and by others, to have proceeded by extension from those of the liver and diaphragm.

These are *all* the cases of pericarditis into the causes of which I have had an opportunity of making *personal* inquiry. They are thirty-one in number (exclusive of slighter cases), and the result may be summed up as follows :—

18 were complicated with rheumatism.
9 . . . Bright's disease.
2 *may* have been complicated with Bright's disease : if not, the cause is unknown.
1 was complicated with malformation of the heart and cyanosis.
1 proceeded from the extension of inflammation from a texture with which the pericardium was in contact.

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* Since the remarks in the text were written, I have seen seven additional cases of pericarditis. Of these, two occurred in

There are some circumstances *common* to two out of the four causes just enumerated, and which it may be worth while to advert to.

1. It appears to be an established fact, that in the course of Bright's disease, the function of the kidneys is seriously disturbed, and that there is less urea, and less of other solid matters excreted with the urine, than in health, and a consequent accumulation of urea, if not of other matters in the blood. 2. There is also in such cases a great proneness to the occurrence of inflammation in various internal organs (vide. p. 543); and this proneness seems to bear a proportion to the imperfect action of the kid-

acute rheumatism, one with Bright's disease and pleurisy, and one with empyema of the left side, and *some* renal disease. In this case the appearances proved the empyema to be of longer standing than the pericarditis. A fourth had well-marked chronic inflammation of both kidneys, with a diminution of their volume, and also with malignant disease of the bladder. A fifth had *some* renal disease, but probably not Bright's disease.

The sixth case may or may not constitute an exception to the remarks upon the causes of pericarditis, made in this communication. It occurred in a delicate boy, the subject of old disease of the mitral valve, and of considerable enlargement of the heart. There was no evidence, during life, of renal disease. There were pains in the limbs, but no swelling or redness of the joints, nor other proof of *acute* rheumatism. There was pneumonia in the upper lobes of both lungs, and pleurisy on the right side. The pleurisy appeared *after* the pericarditis, but whether the pneumonia did or not is uncertain. There was evidence of *some* disease producing condensation of the upper lobes of the lungs, especially of the right, *before* the pericarditis appeared, but the *proof* that *pneumonia* existed, was posterior to the seizure of the heart. The boy died, but no examination of the body could be obtained.

neys, and the consequent imperfect depuration of the blood (p. 563). 3. The internal inflammations in these cases seem to be generally and justly referred to the morbid condition of the blood, because there is seldom any other assignable cause for them, and because they are often associated with other morbid phenomena similar to those which supervene in animals after extirpation of the kidneys.

Again, in acute rheumatism, there is,—1. A great proneness, not only to the occurrence of pericarditis, but also, as I shall prove (p. 508), of inflammation of various other internal parts, especially the lungs and pleura. 2. I shall presently adduce facts to show that the rheumatism is not the *cause* of these internal inflammations, because *they* may appear *first* and the *rheumatism* afterwards, and also because there is no general relation between the severity of the two, either of them being in some cases very trifling, whilst the other is very severe. 3. There is seldom to be discovered *any external exciting cause* of the inflammation complicating an attack of rheumatism. 4. In the number of parts attacked, and in the mobility of the affection, in its comparatively unmanageable character, as well as in other points of its history, there seems to be a great difference between rheumatic and common inflammation of the joints. 5. The peculiarities just mentioned, together with the profuse sour perspirations, the character of the urine and of the blood, the occasional occurrence of rheumatic fever without arthritis, the tendency of symmetrical parts to suffer together, (more

especially observed however in the chronic form of rheumatism,) and other facts which I need not now enumerate, seem to assimilate rheumatic fever to the class of diseases which are acknowledged to depend upon a morbid condition of the blood.—(Vide p. 520, note.)

The facts I have referred to seem to render it highly probable that the immediate cause of the pericarditis and other internal inflammations, which so often complicate Bright's disease and acute rheumatism, as well as of the articular affection itself in rheumatism, is a morbid con tion of the blood.

The case of pericarditis complicated with cyanosis seems rather to support than to invalidate the conclusion I have just arrived at, seeing that the blood is manifestly in an unnatural state in this disease.

Whether the morbid state of the blood be *essentially* the *same* in rheumatism and in Bright's disease, we have no sufficiently precise knowledge to enable us to decide: but in many respects the blood in these diseases obviously differs.

In Bright's disease we *know* there is an excess of urea in the blood, to the influence of which the internal inflammations have been generally ascribed. In many cases of rheumatic fever examined in University College Hospital, there has been found a very considerable excess of urea in the *urine*, especially in the earlier and more active periods of the disease. There *may* therefore be an excess of urea, and perhaps also of other irritating principles, in the *blood*, (from which they pass in excess into the urine,)

leading to some results similar to, and to others different from, those which we observe in Bright's disease.* There are, further, some points of resem-

* The test of this excess of urea was the formation of abundant crystals of nitrate of urea, by the addition of a considerable proportion of nitric acid to the urine, in the state of dilution in which it was voided. In some of the cases (although, from the great abundance of the crystals, I am persuaded not in all) this excess of urea may have been *apparent* only and dependent on a diminution of the proportion of *water* in the urine, in this as in most inflammatory diseases. Becquerel, who has well exposed this source of fallacy, (Sémeiotique des Urines, Paris, 1841, p. 30 to 42,) has also found the total quantity of urea excreted in the urine, *in twenty-four hours*, to be much less in acute rheumatism, and in some other inflammatory diseases, than in health. He further states that the quantity of urea contained in the urine passed in the whole twenty-four hours, very rarely exceeds the physiological limits; it may do so in some cases of polydypsia, or in cases in which the urine is very abundant. He has never observed this increase, and he thinks that if it do exist it is very rare. This result would equally accord with the view taken in the text. If the quantity of urea voided in the twenty-four hours be *increased*, the increase may *arise from* an excess of this principle in the blood: and if the quantity voided be *diminished*, this circumstance (as perhaps in Bright's disease) may *cause* an *accumulation* and a consequent *excess* of urea in the blood.

A more accurate chemical examination of the blood, and of the urinary and other secretions in various diseases, is much to be desired, and seems calculated to yield results of great importance in pathology and practical medicine. I have observed a great excess of urea in the urine (judging by the test above given) in diseases of very various character; especially I think in acute rheumatism, erysipelas of the face, pneumonia, and some other febrile diseases, and also in diseases quite unaccompanied by fever, e. g. in *lepra* in one or two individuals apparently otherwise in perfect health, in internal cancer, in dyspepsia, and even occasion-

blance in the *causes* of rheumatism and of Bright's disease. 1. The only *known* external *exciting* cause of rheumatism is cold, or cold and damp, and this is one of the most common and most influential causes of Bright's disease. 2. In very many cases of rheumatism (a large proportion even) no immediate *exciting* cause can be discovered, and I fully adopt the opinion of Chomel and of others, who believe that in general the exciting cause is the least important of those concerned in the production of the disease; often only the last link in a long chain. I believe the really efficient cause to be such a change in the constitution of the body, chiefly perhaps of the fluids, as is more or less slowly induced under the influence of circumstances unfavourable to health, but often operating imperceptibly, and discovered only by very careful investigation. I allude more

ally in anæmia. Its presence may often be *suspected*, by finding the urine of high sp. gr. at or above 1030 without its containing any sediment, or being deep-coloured, or exhibiting other obvious indications of the presence of other foreign matters in excess, or of a deficient proportion of water.

There can be no doubt that the function of the kidneys and the composition of the urine, experience important modifications in acute rheumatism. I remember the case of a man ætat. 57, admitted into University College Hospital, under the care of my colleague Dr. A. T. Thomson, affected with acute rheumatism. The urine was not albuminous on his admission, but in a short time it became so highly charged with this principle as entirely to assume a solid form on being heated, and its specific gravity at the same time mounted up to 1050. After a few more days, no trace of albumen remained, and the urine had assumed the ordinary appearance and density.

particularly to improprieties—not necessarily gross ones—in diet, clothing, and labour, and to undue habitual exposure to cold or damp, or anxiety of mind, as well as to other agencies which gradually undermine the health. The circumstances in which Bright's disease appears are very similar; those which have appeared to me to be especially influential, being exposure to cold and wet, inadequate clothing, and intemperance. In the chronic form of the disease, which is by far the most common, we rarely discover any immediate exciting cause. The agencies chiefly concerned in its production are those commonly classed under the head of predisposing causes of disease.

Circumstances likely to lead to an imperfect performance of the functions of the skin, would appear then to have a powerful influence in the production both of rheumatism and of Bright's disease; and in both, the functions of the kidneys are materially disturbed. In connection with these facts it may be fairly, I think, assumed that in persons affected with *cyanosis*, the functions of the skin are imperfectly performed, and are more likely than in healthy subjects, to be further interrupted by slight external circumstances.

2. *Of the causes of the pericarditis in cases of old adhesion of the pericardium.*

Having stated what were the causes of the disease actually observed in cases of recent pericarditis, I propose now to inquire into the frequency of the

same causes in cases of adhesion of the pericardium found after death.

Among my notes of *post mortem* inspections, I have found twenty-two examples of old adhesion of the pericardium. Of these, *two* have been considered with the cases of acute pericarditis: there remain therefore for my present purpose twenty cases.

Of these twenty patients, I found that

1 had Bright's disease, [illegible] had never had rheumatismus acutus.

4 had *probably* Bright's [illegible]isease, and *certainly some renal* disease, but had not had rheumatismus a[illegible]s.

4 had Bright's dis[illegible] and had previously had rheum[illegible]n [illegible]cutus.

1 had *some* renal disease, and had previously had rheumatismus acutus.

1 had some renal disease, and had *not* previously had rheumatismus acutus.

5 cases do not furnish any information about either the kidneys or rheumatism.

20

Thus in every case in which any information was given upon the subject, there had previously been acute rheumatism, or there was found either Bright's disease or *some disease* of the kidneys. There are *five* cases in which there was some renal disease, but I think probably not Bright's disease, and no previous rheumatism; and in all of these there were old

adhesions of one or both pleuræ. In three of them the pleura pulmonalis and pleura costalis were adherent on both sides, and both again to the pericardium. In one, the left pleuræ adhered to each other and to the pericardium; and in one the adhesions were between the *right* pleuræ. In the last case, it is not said whether the pleuræ and pericardium adhered, but the adhesions of the *pericardium* itself were limited to the portion covering the *right* auricle.

In five other cases there is no information given about the existence of disease of the kidneys, or of rheumatism. In two of these, there were old adhesions of both pleuræ, (it is not stated whether of the pericardium and pleuræ also,) and in one of these two there were caries of the spine, psoas abscess, and old adhesions between the liver and diaphragm. In the three of these five cases remaining, there were no adhesions of either pleura.

Of the twenty cases of adhesion of the pericardium, therefore, there are *three only* in which the previous existence of pleurisy, or of rheumatism, or the *actual* or *probable* presence of Bright's disease, was not *ascertained*, and the *absence* of all these diseases was *ascertained* in *no one case*. With one exception, the adhesions of the pleuræ were either *double*, or on the *left* side; and in this exceptional case the adhesion of the pericardium was over the *right* auricle only. The pleuritic adhesions were almost always very extensive and thick (in some cases exceedingly so; in two, e. g. *one inch* thick

[illegible] In four out of the seven cases of adherent pleura, the pleura adhered to the external surface of the pericardium also; in the other three it is not said whether the pleura and pericardium were adherent or not. There were adhesions of the pleura in some of those cases also in which there had been rheumatismus acutus, or Bright's disease, or both; but the proportion of these I have not at present ascertained.

The conclusions respecting the causes of pericarditis which were deduced from an examination of the recent examples of the disease, are therefore strongly corroborated by this corresponding examination of the cases of old adhesion of the pericardium. The actual or possible existence, now or formerly, of rheumatismus acutus, or Bright's disease, or pleurisy, has been proved in every case.*

The proportion of cases of old adhesion of the pericardium, in which pleurisy appears to have been the probable cause of the inflammation, is greater, both *absolutely* and also relatively to the other two causes inquired into, (viz., probably five, and possibly seven out of twenty,) than in the cases of recent pericarditis. This may be explained on the very probable supposition, that of the cases of

* In a later part of this paper (p. 544, and Note to p. 549), some reasons have been given for thinking that Bright's disease may have little to do with the production of the *former* attacks of pericarditis, of which the old adhesions are the results. The cases of old adhesion of the pericardium have, therefore, been re-examined, in order to ascertain in how many of them the other two causes above-named may have been present. The following is the result: Number of cases of old adhesion of the pericardium, twenty-two;

pericarditis produced by Bright's disease, and by acute rheumatism, a larger proportion prove fatal in the *acute* stage, than of those due to pleurisy. The cases not proving fatal, i. e., *all* those leading to *adhesions*, must on this assumption be due in greater proportion to the last, than to the two first of the three causes under consideration.

3.—*Of the causes of the pericarditis in cases of white spots on the pericardium.*

I shall now examine into the frequency with which the same causes were present in examples of

of these, six were subject to rheumatism, three had never had rheumatism. In one, no information could be *obtained*, and in twelve no information has been given.

Of the same twenty-two cases of adhesion, there were found old adhesions of the pleura in eighteen, and the state of the pleura is not mentioned in four. Of the eighteen patients having adhesions of the pleura, four had also been subject to rheumatism. The pleuritic adhesions were found on both sides of the chest in ten cases: on the right only in three, on the left only in four, and in one case no information on this point is given.

The result, then, as it regards the two causes of pericarditis under immediate consideration, in this place, is as follows:—

Of twenty-two cases of old adhesion of the pericardium,

In 6 cases there had been previous attacks of rheumatism.
— 14 cases there were old adhesions of the pleura, and there had been no previous rheumatism (besides four in which there had previously been rheumatism).
— 2 cases the state of the pleuræ is *not mentioned*, and there — had been no rheumatism.
22

In two cases *only*, therefore, *could* one or other of these causes have been absent, and in *no case* was both causes proved to have been absent.

white spots on the pericardium without adhesions. The cases of this description which I have collected, are eighty-three in number.

Of these,—

20 { sixteen had previously suffered from acute, and *four* from chronic rheumatism.

10 were found *distinctly* to have Bright's disease.

12 were found *probably* to have Bright's disease.

24 were found to have some disease of the kidneys, the nature of which was not always known.

66

If we deduct from these sixty-six cases, six in which there were at the same time both rheumatism and Bright's disease, and two in which there were rheumatism and *some* renal disease, there remain fifty-eight (out of eighty-three) in which there was found either Bright's disease, or some other renal disease, or in which there had formerly existed acute or chronic rheumatism.

In twenty-three cases there is no information given respecting either rheumatism or renal disease.

In the *two* remaining cases only was the *absence*, *both* of rheumatism and of Bright's disease, *ascertained*, and even in these there is room for doubt, and both of them had old adhesions of the right pleuræ.

Exclusive of those cases in which, either the actual or probable presence of Bright's disease, as

well as of those in which the previous existence of rheumatism, was ascertained, and in many of which there were also old adhesions of the pleuræ, I have examined in how many of the remaining cases old adhesions of the pleuræ were found; and the following is the result :—

1st. Cases of white patch on the pericardium, with no previous rheumatism, but with *some* disease of the kidneys, twenty-two. Of these,

17 had old adhesions of the pleura, viz., four of the right, four of the left, and nine of both.

2[3] had *no* old adhesions of the pleura, but one of them had *cancer* of the pleura.

3[4] had *no* adhesions of the pleura *mentioned*. *One* of them had tubercles in both lungs, and *one* had delirium tremens and gangrene of the lungs.

22

2nd. Cases of white patch on the pericardium, in which no information was given about the existence of rheumatism, or disease of the kidneys, twenty-three. Of these,

11 had old adhesions of the pleuræ, viz., on the right side, one; left, four; both sides, five; and the side not mentioned, one.

6[1] had no old adhesions (one a case of fever, the other of erysipelas), and in

6[2] no adhesions are *mentioned* (one a drinker, and one having tuberculous cavities in both lungs).

23

The following summary of the facts just detailed, may render their import more easy to be understood.

1. In *all* the cases in which I have found the white spots referred to, there MAY have been either Bright's disease, or rheumatism, or inflammation of the pleura.

2. In 17 cases only (viz., $6^{(1)}$, $6^{(2)}$, $2^{(3)}$, $3^{(4)}$,*) has the existence of one or other of these affections not been *actually ascertained* from the histories, and even of these, *five* were *found* to have *some* renal disease.†

3. Of the cases in which neither Bright's disease nor rheumatism was *proved* to exist, or to have existed (but in *all* of which, one, or both of them, *may* have existed), eight only were *ascertained* to have no adhesions of the pleuræ, and even in *two* of these *eight*, *some* renal disease was found.

Thus one or other of the three causes under discussion was *ascertained* to exist, or to have existed, in almost every case examined.

* These figures (1) (2) (3) (4) refer to corresponding figures on the last page.

† Among these patients, several others were the subjects of diseases, whose nature and effects are such, that the cases may be adduced in support of the general conclusions, which it is the object of this paper to establish,—e. g., two had tuberculous cavities in both lungs, (and probably therefore pleuritic adhesions,) one had cancer of the pleura, one gangrene of the lungs and delirium tremens, one fever, one erysipelas, and one was a drunkard. These, with the five having *some* renal disease, and noticed above, comprise twelve out of the seventeen cases cited in the text.

As I may not again have occasion to speak in detail of pleurisy, the present may be a fitting place in which to recapitulate my reasons for considering this disease to be *really* a cause of pericarditis.

1. It may be assumed as an ascertained fact, that inflammation does extend from one portion of a tissue to another portion of the same, or to another tissue, (especially of the same anatomical species, e. g., from *one serous* membrane to *another serous* membrane,) in circumstances in which no other cause for the extension can be discovered, except the contact of the part first inflamed with that which subsequently becomes inflamed.

2. In one of the cases of *acute* pericarditis which I have quoted, (and *probably* in another also which I have since seen,) and in several of the cases of adherent pericardium, *no other cause* but this could be discovered.

3. Out of twenty cases of old adhesion of the pericardium, there were seven in which the inflammation producing the adhesion was believed to have proceeded by extension from the pleura. In support of this opinion, I would remark,—(*a.*) That there were adhesions of the *left* pleura in *six* cases, (both pleuræ in five, left one, right one,) and according to my *recollection*, (for I have not *counted* the cases,) the cardiac *adhesions* affected either the *whole* pericardium, or that portion covering the *left* cavities more particularly. In the *single* case in which the adhesions of the pleura were limited to the *right* side, the adhesion of the

pericardium was limited also to the portion covering the *right* auricle. (*b.*) In four out of the seven cases, the pleura adhered to the outer surface of the pericardium also: in three, no information is given on this point.

4. Of twenty-eight cases in which inflammation of the pleura was the only *obvious* cause of the inflammation leading to white spots on the pericardium, the *adhesions* of the pleura were on the *right* side in nineteen, on the left alone in eight, and the side is doubtful in one case: it is also a well-known fact, that the white spots are more frequently found on the pericardium covering the *right* cavities of the heart than *elsewhere*.

Old adhesions of the pleuræ were found in many cases besides those enumerated. They have been counted in those cases of old pericarditis *only*, in which neither rheumatism nor Bright's disease was *ascertained* to have been present.* In *some* of the cases in which adhesions, both of the pericardium and of the pleuræ, were found, the inflammation may have extended from the former to the latter organ; in *other* cases again, the two inflammations may have derived their origin from a *common* cause, and whether beginning *simultaneously* or at *different times*, may have been independent of each other. There can be little doubt however, that in a third class of cases (and those *may* be the most numerous) the inflammation has extended from the *pleura* to the *pericardium*; because the *post mortem* appear-

* Vide Note to p. 466.

ances sometimes *show* the disease in the former texture to be the *older* of the two, and because also, according to the facts adduced in another part of this communication, (p. 537 and 541,) pleuritis is a much more common disease than pericarditis.

Before terminating these general remarks on the causes of pericarditis, I would desire to guard the members of the Society against any misconception of the meaning of my statements, by reminding them what has been the especial object of my inquiries up to this point. I have first examined all the cases of acute pericarditis which have fallen under my observation, with a view to discover what were the causes to which the disease *in those cases* might, with most probability, be referred. I have next endeavoured to establish the *reality* of the influence of the causes *so observed*, and to obtain some *measure* of their importance, by inquiring how often they had been present, and might therefore have operated, in those cases in which, after death, pericarditis was found to have existed at some former period. I have thus *limited my inquiries* to the three chief causes already considered, but I do not wish to be understood to assert that there are no other causes of the disease. I believe that other causes do exist, and it is possible that some of them may have been in operation in the cases of old adhesion of the pericardium; but as the three causes I have treated of were ascertained to have been *actually* present in *most*, and to have been *possibly* present in *all* the cases which I have examined, it seems reasonable to

conclude that these are the most *common*, and consequently the most *important*, causes of the disease.

It may not be uninteresting to compare the results which have been stated, with those arrived at by some other observers.

1. *Of the causes of pericarditis in general.*

Louis* states, that the exciting causes of pericarditis are unknown in the greater number of cases. The authors to whom he had referred, mention them only in two subjects. One† of these had received a violent blow with the fist on the pericardial region, fifteen days before the symptoms appeared; the other‡ had ridden hard on horseback during several successive days. Louis appears to have found an exciting cause in *one* only of the *seven* cases observed by himself. The patient, a female, was seized with the disease, after experiencing great grief, and at the end of a long journey on foot (210 leagues in twenty days). He likewise *infers*, from the frequent complication of pericarditis with pleurisy and pneumonia, the probability of the former disease arising from the same causes as the two latter, and also being influenced by them.

Bouillaud§ treats very lightly the opinion of Louis, that the causes are in most cases unknown. He admits, that in *some cases* they cannot be ascertained; but his statements *imply* that these are only exceptional instances. The *great* cause, in his opinion, is exposure to cold after copious perspira-

* Recherches Anatomico-Pathol., p. 293.

† Vide Corvisart. ‡ Vide Bertin.

§ Traité Clin. des Malad. du Cœur, 1ere ed., t. i., p. 470-1-4.

tion and fatiguing labour; and he seems to speak of this cause, and of acute rheumatism, almost as if he considered them to be the same thing.

M. Hache,* however, on analysing Bouillaud's own cases, has drawn from them a different conclusion. Of thirty-six cases reported in the work referred to, the causes appear to have been *unknown* in twenty-two (nearly two-thirds), or at least none are mentioned; and in *six* cases only did a *manifest* cause exist, as exposure to cold, at a period near the beginning of the attack. Of the eight patients whose cases are reported, by M. Hache himself, two had been exposed to cold whilst perspiring, and had also rheumatismus acutus. In four, no exciting cause was discovered, but only *remote* causes more or less probable; and in two there was no known cause of any kind.

2. *Of rheumatism.*

Of all the causes of pericarditis, acute rheumatism is that which has attracted by far the greatest share of attention, and is generally considered to be incomparably the most frequent. So strong is the hold which this opinion has obtained upon the minds of medical men, that I have very frequently heard them express their surprise on meeting with a case of disease of the heart which had originated independently of rheumatism.

Dr. Hope,† referring to the causes of pericarditis,

* Memoire sur la Péricardite. Archiv. Gen. de Medicine, 2e Série, t. ix., 1835, pp. 325, 330-1.

† On Diseases of the Heart, 3rd ed., p. 177.

says, "and far above all acute rheumatism;" and referring to acute, or sub-acute rheumatism, he describes it as "an affection, which, whether severe or mild, whether in its early or later stages, is, beyond comparison, the most frequent cause of pericarditis and endocarditis."* He likewise states that "acute rheumatism had preceded in about three-fourths of the worst cases of valvular disease, and adhesion of the pericardium, which have occurred amongst upwards of 10,000 hospital patients," whom he had treated during the preceding four and a half years.†

Dr. Watson thus expresses himself respecting the same question:—"Acute pericarditis is liable to arise, like all other internal inflammations, after exposure to cold, or when no exciting cause is to be discovered. But for one such case of what may be called spontaneous acute inflammation of that membrane, you will meet with a dozen, or more, in which it occurs in connection with a disease that we have not yet had before us—acute rheumatism."‡

Dr. C. J. B. Williams considers that "of the causes of pericarditis, rheumatism is by far the most common; but it still more frequently produces endocarditis."§

3. *Of Bright's disease.*

Pericarditis has been mentioned by various writers as one among the secondary inflammations to which Bright's disease gives rise. But the great frequency

* Op. cit., p. 161. † P. 178.

‡ Lectures, vol. ii., p. 263.

§ Lond. Med. Gaz., vol. ii., 1837-8, p. 866.

of Bright's disease as a complication of pericarditis, and its great frequency relatively to rheumatism, and to the other causes of this inflammation, appear never to have been suspected.

The strongest statement which I have met with respecting the occurrence of pericarditis in connection with Bright's disease occurs in the original paper of Dr. Bright himself. "Out of seventeen dissections, we have found ten or eleven betraying inflammation of the pleura, generally old, but sometimes of more recent date. We have found three instances in which the patients had suffered decided attacks of inflammation in the pericardium, shortly before death; and in two of these cases we had proof of some previous affection of the same kind. In one only were the signs of inflammation in the peritoneum well marked. Five out of the seventeen had altogether escaped inflammatory affections of the serous membranes."* (See also Table II. p. 539 of this communication.) The prominent reflection in the mind of Dr. Bright appears to have had reference to the great frequency of internal inflammations, as a consequence of renal disease. To this he was naturally led by the course of his investigations. But his inquiries did not lead him to consider, nor does he appear to have suspected, the *great proportion* of all cases of pericarditis which arise from renal disease.

Dr. Christison, in reference to the secondary affections in Bright's disease, observes, that "pleurisy

* Reports of Medical Cases, 1827, vol. i., p. 71.

is the most frequent of the serous inflammations; peritonitis is, upon the whole, rare; pericarditis is also seldom seen."*

Drs. Rilliet and Barthez, in treating of Bright's disease, as it occurs in children, state, that "the principal complications are inflammation of the same organs in which dropsy is liable to appear. The most frequent of all, are certainly inflammations of the serous membranes, then of the lung. . . . . The inflammations of the serous membranes are pleurisy, peritonitis, and much more rarely pericarditis."†

The chief particulars, therefore, in which my results differ from those obtained by other observers, are these:—1. I have found an adequate cause for the pericarditis in *all* my cases. 2. Whilst I have found rheumatism, as a complication, in quite as great a proportion of cases as any one who has given us a numerical account of his observations, yet its frequency, relatively to other causes, has been less in my experience than in that of any writer with whom I am acquainted. 3. I have found Bright's disease to be the cause of pericarditis in a large proportion of the cases which have fallen under my notice.‡

* On Granular Degeneration of the Kidneys. Edinb., 1839, p. 90.

† Traité Clinique et Pratique des Maladies des Enfans. Paris, 1843. Vol. i., p. 603.

‡ Since this communication was sent to the Society, and before it was read, the "Lectures on subjects connected with Clinical Medicine, comprising diseases of the heart," by Dr. Latham,

Having taken a *general* view of the causes of pericarditis actually observed in my cases, I propose now to inquire, more particularly, into some of the circumstances connected with two of these causes.

I.—OF RHEUMATISM, CONSIDERED AS A CAUSE OF PERICARDITIS.

1.—*Frequency with which pericarditis and endocarditis occur in acute rheumatism.*—We have seen the importance of acute rheumatism as a cause of pericarditis, in the fact that this cause existed in two-thirds of the cases examined. I shall now endeavour to ascertain what amount of confirmation the conclusion thus arrived at, may receive, from an inquiry into the frequency with which pericarditis occurs in the course of rheumatism.

have been published. The following extract from this valuable work will show how little the author suspects, and probably, therefore, how little physicians generally suspect, the important part that Bright's disease plays in the production of pericarditis. "My own experience of pericarditis is mainly derived from what it is as an accompaniment of acute rheumatism. I have seen the disease, indeed, under other circumstances; but it has been very seldom; so seldom, indeed, that I have little acquaintance with other conditions, external or internal, conducing to it. I can neither tell whence to look for it, nor when to expect it, except when it occurs as a part of acute rheumatism."

"The pericarditis which is acute and rapidly progressive, and, unless arrested by timely and effectual treatment, full of peril, this is the pericarditis I mean. And with this the practice of a large hospital has rendered me familiar, by presenting me every year with numerous instances of it in alliance with acute rheumatism. But, separate from acute rheumatism, even the practice of a large hospital does not present me with more than an instance or two of it in several years."—P. 136.

With reference to this question, I examined, several years ago, a number of cases of acute rheumatism, occurring in University College Hospital. The greater number of these patients were treated by Dr. Elliotson; but a few by Drs. Carswell and Williams. The results are as follows:—

Of forty-seven cases of acute, or sub-acute, rheumatism, *taken indiscriminately*, I found that

In	11	(nearly one-fourth) there was valvular disease, either old or recent, but probably in most of the cases old. Some of these patients had also hypertrophy of the heart, and some had not.
In	2	there were signs of slight valvular disease, probably from actually existing endocarditis in one, if not in both of them.
In	25	(more than half) there was no evidence of the existence of any kind of disease of the heart.
In	9	the state of the heart is not mentioned, and I conclude it was healthy in most of them, because it was the custom, both of the physicians and of myself, to examine the heart in all cases of rheumatism.
	47	

Of an otherseries* of eighty-six cases of acute

* *Some* of the preceding forty-seven cases are included in these eighty-six; how many, however, my notes do not enable me, without some difficulty, to determine.

or sub-acute rheumatism, also *taken indiscriminately*, I found that

In	5	there was acute pericarditis (in *one* of these there was also acute endocarditis).
In	19	(less than one-fourth) there was valvular disease, either old or recent, but probably in most of the cases old. Some of these patients had also hypertrophy of the heart, and some had not.
In	19	there was no evidence of disease of the heart of any kind.
In	43	the state of the heart is not mentioned, and I conclude that this organ was healthy in most of them, for the reason stated above.
	86	

The proportion of cases of acute pericarditis, therefore, was one in seventeen; and of disease of the heart of some description, but chiefly old disease, about one in three and a half. The heart was probably free from any readily appreciable disease, whether old or recent, in more than two-thirds of these cases of acute rheumatism.

I shall now examine the cases of acute rheumatism treated by myself in University College Hospital, in the course of three years and a quarter; and, in order to render the results applicable to some other purposes, I shall give a separate account of the cases treated in each year.*

* The cases for the first year are so much fewer than those of

First period of twelve months—1841-2.

Number of cases of acute or sub-acute rheumatism, *nine*. Of these patients, two had slight morbus cordis, old or recent; five had no sign of disease of the heart; and in two, the heart is not *mentioned*, and was probably healthy. There was no case of pericarditis.

Second period of twelve months—1842-3.

Number of cases of acute or sub-acute rheumatism, *twenty-four*. Of these patients, one had acute pericarditis; ten others had some kind of morbus cordis (there was valvular disease of old standing in two of them, and in eight it could not be determined whether the valvular disease were altogether old or recent). In ten, the heart was healthy; and in three, its condition is not mentioned, and was probably, therefore, healthy.

Third period, of fifteen months—1843-4.

Number of cases of acute or sub-acute rheumatism, *forty-two*. Of these patients,

24	had some kind of morbus cordis.
14	had no morbus cordis; and in
4	the heart is not mentioned, and was probably
—	healthy.
42	

the second and third years, that I have no doubt some must have been omitted from the tables which were prepared at the end of each session, and from which the calculations were made at the time. There has, however, been no *selection*, and a case of rheumatism, complicated with pericarditis, would certainly have been much less likely to escape notice than one not so complicated.

Of the twenty-four having morbus cordis, eighteen had slight valvular disease (generally *very slight*). This disease was probably of old standing in most of them, and was not *known* to be *recent* in any. Three had a little hypertrophy. There was well-marked acute pericarditis of considerable severity in *five*, *very slight* pericarditis in two more, and in *two* others, the same disease might be present, but in so trifling a degree, that its existence was doubtful. Two of the cases of pericarditis were complicated with well-marked endocarditis, and in two others the same disease might be present, but its existence was doubtful.

All the five severer cases of pericarditis occurred between the 1st April and 5th Oct. 1844; and it was remarked at the time (and the remark is confirmed by the figures already given), that the disease was observed much more frequently than *usual*, within the period referred to.

Summary.—The cases of acute or sub-acute rheumatism treated by me were seventy-five in number. Of these, thirty-seven, or one-half, had morbus cordis of some kind. The rest had probably no morbus cordis, for twenty-nine are *stated* to have had none; and in *nine*, no mention is made of the heart.

Of the cases having some morbus cordis, *six* had *acute pericarditis* of considerable severity, *two* in a very slight degree, and in two its existence was doubtful.

There was valvular disease, either old or recent, in thirty cases. In two others there was valvular

disease *known* to be recent, and in two more there was doubt whether it was recent or not. The four cases last named were complicated with pericarditis. The *proportion* of cases of pericarditis, then, (excluding the two very slight and two doubtful ones,) was one in twelve and a half; and of disease of the heart of some kind, but chiefly of old standing, about one in two. The heart was healthy in one half.

The *frequency* of pericarditis, and of morbus cordis generally, as observed in my cases, was considerably greater than in the cases treated by the other physicians conjointly. The difference may, in part, perhaps, be owing to a progressive increase in the amount of attention paid to the heart, and to a consequent habit of *recording slighter* cases of morbus cordis than, at an earlier period, might be thought *worth* mentioning; and of such, indeed, as I now think are subjects of curiosity rather than of much practical importance.

The question of the frequency of pericarditis arising in the course of acute rheumatism has excited much discussion within the last few years, and has elicited much difference of opinion from high authorities; it may, therefore, be worth while, in this place, to refer to some of the results which have been arrived at.

Bouillaud, who has contributed more than any other person to excite the discussion of this question, stated, in 1835, that " pericarditis exists in about one half of the individuals affected with violent acute

articular rheumatism."* In 1836, he says he had found that acute rheumatism affecting several articulations had been accompanied, in eight cases out of nine, by rheumatism of the sero-fibrous tissue of the heart.† In a still later publication, in 1840, he makes the following statement:—" In 114 cases of acute articular rheumatism, of which we have made the most exact resumé, there occurred seventy-four of great or of medium intensity, and forty of a slighter description. Now, among the seventy-four cases of the first category, we have counted sixty-four in which the coincidence of endocarditis, or of endo-pericarditis, was *certain*, and three others in which it was *doubtful*; whilst among the *forty* cases of the second category, there is but *one* in which the coincidence in question has been ascertained to exist."‡ Hence this writer concludes—1. " That, in violent acute articular rheumatism generally diffused (généralisé), the coincidence of endocarditis, of pericarditis, or of endo-pericarditis, is *the rule*, and the non-coincidence *the exception*."

2. " That in acute articular rheumatism, which is slight, partial, and apyretic, the non-coincidence of endocarditis, of pericarditis, or of endo-pericarditis, is the rule, and the coincidence the exception."§

* Traité Clin. des Malad. du Cœur., Pref., p. 14.

† Nouvelles Recherches sur le Rhumatisme, artic. aigu. p. 27.

‡ Traité Clin. du Rhumat., &c. Pref., p. 12.

§ Traité Clin. du Rhumat., &c., 1840, p. 12.

Bouillaud's cases of "great and medium" intensity, are probably the same as those I have called *acute* and *sub-acute*; for he characterises the slighter (légers) cases by the expressions, "rhumatisme articulaire aigu, leger, partiel, apyretique."

Dr. Hope, after quoting the opinion of Bouillaud, that *one-half* of the individuals affected with acute rheumatism have symptoms of pericarditis, or of endocarditis, and often of both, makes the following remark upon it: "According to my own observation, the statement is not wide of the truth, when rheumatic affections are neglected or inefficiently treated:" * but in the next page of his work, referring to the occurrence of "inflammation of the heart" in acute rheumatism, treated in the manner which he there advocates, he adds, "I think that about one case in twelve would be the maximum in my practice."

Dr. C. J. B. Williams expresses his opinion in the following terms: "I can confidently state, that I have found signs of endocarditis, or pericarditis, or both, to a greater or less extent, in fully three-fourths of the cases of rheumatism which I have examined in the last three years." †

Dr. Macleod, speaking of "rheumatic fever," or the fibrous form of acute rheumatism, states that, "Of the 226 cases of acute rheumatism, above alluded to, the heart was affected in fifty-two, or rather more than one-fifth; but taking the whole

* On Diseases of the Heart. 3rd edit., p. 178.

† Lectures in Lond. Med. Gaz., 1837-8. Vol. ii., p. 866.

number of cases of rheumatism, including every form, the proportion of such complication I believe to be little more than half the above."*

Dr. W. Budd found that rheumatism of the heart came on in one half of forty-two cases of rheumatism. He also found "that rheumatic inflammation of the pericardium is less frequent than that of the valves of the heart." "In twenty-one cases of acute rheumatism affecting the heart, which were taken indiscriminately, and whose history was written with exactness, there were five only of pericarditis, and in all these the lining membrane was also affected."†

Drs. Rilliet and Barthez observed acute pericarditis four times in eleven cases of articular rheumatism (*acute*, I believe).‡ These cases, it is to be remembered, occurred amongst children, and in the children's hospital in Paris.

M. Chomel in the resumé of his practice during the season of 1834-5, refers to forty-nine cases of acute articular rheumatism carefully observed, in which no example was found of pericarditis or of endocarditis.§

In the spring of 1841, I heard the statement of the result of his public practice delivered by Chomel at the end of his course of clinical lectures, and I made the following memoranda which relate to

* On Rheumatism, &c. London, 1842, p. 154.

† Library of Medicine, vol. v., pp. 200 and 194.

‡ Traité Clinique et Pratique des Maladies des Enfans. Paris, 1843. Vol. i., p. 210.

§ Quoted by Bouillaud in Traité Clinique, 1840, p. 133.

this subject:—In a period of ten months, he had treated in the Hôtel Dieu in Paris, thirty-one cases of acute rheumatism; of these, nineteen were very acute, and twelve sub-acute. Among these patients, *one* only had had symptoms of disease of the heart, (palpitations,) and in one case only, the rheumatism was complicated with disease of the heart, ***beginning whilst the patient was in the hospital.*** Bruit de soufflet was heard in seven cases, and in two others its presence was suspected only.

M. Chomel likewise communicated the following results of his previous experience:—In the course of six years he had treated in hospital practice eighty-six cases of acute rheumatism.* Among these patients were found *six* who had symptoms of disease of the heart before the rheumatic attack, and in four others, disease of the heart supervened in the course of the rheumatism. There were twenty-nine patients (or one-third of the whole) in whom a bruit de soufflet was heard during some days.

In the thirty-one cases referred to, (and *perhaps* in all the eighty-six,) the heart was examined with peculiar care, in consequence of the very acrimonious and personal discussions of the question before us, which [illegible] between some [illegible] cine in Pari[illegible]

* Looking at t[illegible] to *include* the thirty[illegible] spoken of.

† We ought to bear i[illegible]

Of eight cases of acute rheumatism collected with great care by M. Grisolle after the publication of Bouillaud's work in 1836, pericarditis did *not* occur in *one*.*

The reference I have just made to the opinions of various observers, exhibits a very considerable difference in the results at which they have arrived. Upon this difference I beg to make the following observations :—

1. I think we are entitled to receive with some allowance, and certainly with considerable caution, those statements which are not accompanied by a precise and numerical exhibition of the cases upon which they are founded. Unless when so accompanied, we can never know whether the results given are merely *general impressions* left in the mind of the observer, or whether they are such as have been arrived at by an actual *enumeration* of the cases with their complications. Every one at all accustomed to such inquiries, is aware how widely results of the first description are not only *liable*, but even *almost certain*, to deviate from the truth, and that not only when obtained by incompetent individuals, but likewise by those whose accuracy of observation and good faith are unimpeachable.

and gout to be the *same disease*. As the cases above ever, occurred in hospital practice and in Paris, it or nearly all, of them were examples of rheu-

Leçons de Clinique Médicale. Rhu- p. 205.

2. Several of the authorities quoted, have stated their results in reference to both endocarditis and pericarditis, without distinguishing the two: others have either distinguished them, or have furnished us with the requisite data for doing so. If we take pericarditis and endocarditis *together*, we shall find that Bouillaud observed this complication in six-sevenths of the cases of acute rheumatism; Dr. Williams in fully three-fourths; Chomel in about half; Dr. Hope in half (or one-twelfth in rheumatism treated in a particular manner*); and Dr. Budd

* If it could be established that, under different methods of treatment, so great a difference in the results can be obtained, the fact would be most important; but I feel it my duty again to protest against all conclusions drawn from facts so loosely stated. I utterly repudiate such evidence, come from whom it may, in every department of medicine, and, most of all, in that department which is perhaps the most difficult, viz., in which we have to estimate the absolute and relative effects of different plans of treating disease. For anything that appears to the contrary, the opinion quoted in the text is a *mere guess*—it is opposed to the plainest probabilities, and would be more fitly placed among the exaggerations of a caricaturist than among the sober conclusions of an inductive philosopher. Such *guesses* are the bane of therapeutics, and a fruitful source of those remedies and systems which have successively risen above the medical horizon, flourished for their brief day, and then disappeared for ever, unless when resuscitated for an hour under similar influences in some succeeding generation. Some lover of books might do the profession a service by collecting all such crudities into a volume, which might serve as a beacon to our course, whether backwards into the sea of books, or forwards into that sea of nature, on the shore of which Newton, with equal truth, modesty and beauty, described himself as standing. I am half inclined to volunteer my aid to any such labourer,

in half. I found the same complication in half the cases treated by myself, and in one in three and a half of those treated by the other physicians to the hospital conjointly. So far the conclusions arrived at by the different persons referred to (with the exception of Bouillaud) agree to a very considerable extent.

All the writers quoted, excepting Chomel, either *directly state* or seem evidently to *imply* that the cardiac disease in the cases observed by them was *recent*. It is in reference to this question that a considerable difference is apparent between their results and those of Chomel and myself. Chomel found only *one* case in twenty-one in which the cardiac disease was ascertained to have supervened during the progress of the attack of rheumatism under observation. In one-third of his cases, however, he found a bruit de soufflet, which in *most* of them I think *probably* arose from valvular disease, although he does not admit, nor do I assert, this opinion to be proved.

In the cases of acute rheumatism occurring in University College Hospital, there was found to be *recent* endocarditis or pericarditis—

and by way of earnest, I offer him, as a title for his work, "Random Recollections of Medical Experience."

I should be sorry to leave any one under the impression that these remarks are directed against *Dr. Hope*; it is against the system that I raise my voice. Had I been seeking them, I could have found abundant materials in other books for similar comment; such blots are only more conspicuous when found in a work of so much value as that of Dr. Hope.

1. In one in seventeen of those treated by the physicians before named.

2. In one in five, about, of those treated by myself.

I observed the existence of *valvular* disease (*not ascertained* to be recent) in one out of two and a quarter cases of rheumatism, which proportion is nearly the same as that in which Chomel found a bruit de soufflet (viz., one in three).

It thus appears that whilst the proportion of cases of *recent* inflammation of the heart, which I found to complicate acute rheumatism, is *less* by one half than that given by the authors quoted above, it is *three* times *greater* than that deduced from the cases in University College Hospital, which were not treated by me, and *four* times greater than that found by Chomel. It is worthy of remark, that the two results last referred to, very nearly agree.

Causes of this difference of opinion.—The differences in the results which have been quoted are too great to be accidental, and one or two reflections have occurred to me, which I beg to offer, as going some way towards reconciling these discordant statements.

1. The fact that a greater number of cases of recent inflammation was observed by me than by M. Chomel, I would explain by the supposition, (which, although an *assumption*, I believe to be true, because it is founded upon a *personal* observation of his practice,—I make this statement also without forgetting the remark previously made at page 488, and which

may only refer to about one-third of Chomel's cases,) that circumstances led me to examine the heart with more care, and more unvarying regularity than M. Chomel did, and that, in consequence, I have either observed some such cases as he overlooked, or which he did not think of sufficient importance to be recorded. The same supposition may account for the *agreement* in the numbers deduced from the cases in University College Hospital, not treated by me, with the numbers of M. Chomel, as well as for the *difference* between both those numbers and my own.

2. Much of the difference between my results and those of all the other physicians referred to, except Chomel, may be ascribed, I think, to another cause. The gentlemen in question have said nothing about the distinction of *previously existing* valvular disease from that supervening during the attack of rheumatism under observation. Hence as they have omitted all *mention* of examples of old valvular disease in their cases of rheumatism, they must either have included them with the cases of recent inflammation, without distinguishing them, and have spoken of them all as if recent, or they must have remained *intentionally* silent respecting the cases of old standing, for they *could* not fail to *observe* them: which of these views is the more likely to be true, every one will judge for himself. I *believe* the former to be the correct one, and shall proceed upon that supposition.

Now the *practical assumption* (I do not say the

direct statement) which I have ascribed to the writers referred to, that the signs of valvular disease of the heart, observed in cases of acute rheumatism, arise in all instances from *recent inflammation*, is certainly erroneous. In a great number, perhaps in most, of the cases, the heart-disease is of older date than the rheumatism,* and in many of the rest, the duration of the valvular disease is un-

* The truth of this statement will appear from the consideration and comparison of the facts which follow, and of those given at page 503-4. Between December 1837, and February 1841, I examined and noted the condition of the heart in 1,026 patients, in University College Hospital, *taken indiscriminately* from among those admitted for all kinds of diseases. This was done for the purpose of ascertaining what proportion of them had signs of disease of the heart; and I found physical signs of a greater or less amount of disease in this organ in 413 cases (these are exclusive of cases of anæmia), as well as signs more or less doubtful in 67 more cases. They were, for the most part, examples of valvular disease, but include, also, a few of hypertrophy without valvular disease. Of these cases, 105 belonged to the class of morbus cordis; that is to say, the affection of the heart constituted the main disease. As the signs were noted almost always at my first visit after the patient's admission, all those cases of inflammation in the heart are excluded which supervened after that time: and, as I find only *one* case distinguished in which the endocarditis was *known* to be *recent*, and only two of pericarditis (one acute and one chronic), I think the numbers given may represent pretty accurately the proportion of physicians' cases of all kinds—and including, of course, cases of rheumatism—which exhibited signs of *old* disease of the heart. Now, as the proportion of *valvular* diseases in *all* the cases *approximates* to the *proportion* of valvular diseases in the cases of acute rheumatism, it seems to be a necessary conclusion, that most of the examples of valvular disease,

certain; for in *most* instances in which the physical signs are observed when the patient is first ex-

complicating acute rheumatism, are of older date than the rheumatism itself.

The *excess* of cases of valvular disease in the class of acute rheumatism, compared with the class of "all diseases," is probably due to, and therefore represents, the *amount* of the excess of RECENT *endocarditis* in the one class above the other; and, as we have just seen that the class "all diseases," contains in it *hardly any* examples of *recent* disease of the heart, the *same excess* may be taken to represent the ABSOLUTE amount of cases of acute endocarditis supervening in the course of acute rheumatism. It therefore becomes interesting to calculate the amount of the excess referred to, and it will be found to be as follows:—

Of the 413 cases of heart-disease, twenty-eight were unattended by any murmur, and were cases of hypertrophy; and 385 cases exhibited a murmur of greater or less intensity.

Of the 1,026 cases, therefore, 37·524 per cent. were accompanied by a cardiac murmur; or,

We may exclude eighty-two cases of acute or sub-acute rheumatism from the 1,026 cases. Of these eighty-two cases of rheumatism, fifty-five were accompanied with a murmur, and twenty-seven were not.

There remain, therefore, 944 cases without rheumatism; among which are found 330 cases with a valvular murmur, i. e. 35·063 per cent.

We have seen, that among cases of acute rheumatism (pp. 483-4), old or recent valvular disease was found in thirty-four out of seventy-five, or in 45·333 per cent.

The *excess* in the amount of valvular disease in cases of acute rheumatism, therefore, above that in *all diseases indiscriminately*, is 7·809 per cent.; and above that, in *all diseases exclusive of rheumatism*, 10·270 per cent.

The frequency of acute endocarditis in rheumatism thus arrived at, will be made the basis of some future calculations, and also of comparison with the observations of other physicians. It is, therefore, important to keep clearly in view a fundamental differ-

amined, we have no means of distinguishing whether the valvular affection be old or recent.*

ence in the character of the data of those writers who will be quoted, as compared with the data made use of in the above calculations. The writers referred to, estimate the frequency of endocarditis from *direct* observations, made during the life of the patients. I have not *attempted* to do this, because I am persuaded that a number of cases of endocarditis cannot be *distinguished* during life, from cases of *old* disease. (See the note below.)

* On this subject I would remark,—1. That in old valvular disease, and in recent endocarditis, the modifications in the sounds and impulse of the heart, and in the extent of dulness, on percussion, are in very many cases the same. 2. The pyrexia may be a part of the rheumatism, and therefore affords us no aid. 3. In very many cases of both kinds, there is no *pain* in the cardiac region, or the pain is plainly seated in the integuments; and in all cases, taken singly, it is a very uncertain guide. 4. The permanent *disappearance* of the bellows' murmur, I believe to be the exception, instead of the rule, in cases of *recent* endocarditis; and, therefore, it does not generally distinguish this from old valvular disease.

I have been enabled to discover the existence of *recent* endocarditis, in a few cases, chiefly by the following circumstances: 1. By the appearance of a bellows' murmur, which could not be ascribed to venesection, or to any other obvious cause; and in a patient who was found to be free from it on admission. 2. By some considerable change in the rhythm of the heart, in cases in which it was at first found to be natural. I have observed the pulse to become *decidedly less frequent*, and each individual beat to be more slowly performed; in other words, *slow* and *tardy*. The ac[illegible] of the heart become[illegible] force and rapi[illegible] the [illegible] often also interm[illegible] served the bellows [illegible] again become regu[illegible] expected the contracti[illegible]

3. If in a large proportion of the cases of valvular disease referred to, the physical signs *were proved* to arise from, and therefore to indicate, *existing* inflammation, the conclusion, that endocarditis is more frequent than pericarditis, would not yet be warranted. For anatomical and physiological reasons, a very slight amount of endocarditis, having its seat in the valves, will give rise to an appreciable murmur; and it is almost certain that an equally trifling amount of pericarditis would not be so readily detected, and that such cases have not in fact been counted among those spoken of by the writers in question.—(Vide also on this subject, note to p. 551.)

tardy in a disease which must increase the sensibility of its lining membrane. Perhaps the cause is the same as that of suspension of the peristaltic action of the intestines in peritonitis, and as that of the paralysis of the intercostal muscles, which some physicians believe to occur in pleuritis. Be the explanation what it may, however, I am quite sure, both from the careful observation of such cases during life, and from an examination of the heart after death, that the symptoms I have described are produced by endocarditis. But then they are *not* present in *all cases* of endocarditis, as I have also *ascertained*. Were I to trust to the *im*- on my own mind, unchecked by figures, I should say found the symptoms in question, in cases of endo- mpanied by pericarditis; and that in cases of I have found the *frequency*, and perhaps the e increased, with or without intermittent or of pericarditis, and of endo-pericarditis, d quickness of pulse, would appear to of the slowness and tardiness

4. The excessive proportion of cases of heart-disease, observed by Bouillaud in his cases of rheumatism, (nearly twice as high as that found by any other physician,) does not admit of the same explanation; nor can I suggest any other view which appears to me so reasonable as this, viz. either that he has fallen into some error in the *enumeration* of his cases, or that he has unduly increased the number of cases of valvular disease, by including amongst them cases in which there was a bellows' murmur, produced by anæmia, whether idiopathic, or consequent on the large and repeated venesections which he is known to advocate and to practise. Bouillaud's own explanation of the different results obtained by himself and others, I believe, is this—that *they* have *overlooked* cases which *he* has observed and recorded. This explanation is no doubt true to a certain extent, but only in reference to *some* of the individuals to whose opinions he is opposed; and it is therefore totally inadequate to serve the purpose he has in view. As I have myself suggested a similar explanation of the difference, in the results which I obtained from an examination of the cases treated by myself in University College Hospital, and of those treated at an *earlier period*, and by several other physicians *conjointly*, I am anxious to guard against any misunderstanding, by adding a word in further explanation of my meaning. I do not intend to insinuate (what I have heard Bouillaud affirm) that others have overlooked cases which I

have counted, *because they were not* COMPETENT to observe them; but I believe this may have happened *partly* because circumstances have occurred to me which induced me to give more than ordinary attention to this subject; and partly because I may, (for the last-mentioned reason) have *recorded* cases which they perhaps *observed*, but did not consider of sufficient importance to be mentioned. The physical signs of valvular disease do not require for their detection so delicate or so practised an ear, that auscultators of acknowledged respectability have need to resort to the odious expedient of charging each other with an *inability* to appreciate them.

If we consider *acute pericarditis* apart from endocarditis, or valvular disease, the results are as follows. Bouillaud found this complication in one-half of his cases of acute rheumatism. Dr. Macleod, in one-fifth in *adults*, and in one-half in children (four in eight). Rilliet, in one-third in children. Dr. W. Budd found it in one out of every eight and a half cases; and I found it in one out of twelve and a half of *my own* cases, (or, including two *slight* cases, in one in nine and a half,) and in one out of seventeen of the rest of the cases observed in University College Hospital. Upon these statements I would remark:—

1. That Bouillaud gives no *numerical data* for pericarditis alone; hence we cannot feel assured that he has carefully enumerated his facts.

2. Dr. Macleod, whose proportion of cases of pericarditis stands next after Bouillaud's, has made *no mention* of endocarditis, or of valvular disease, and therefore I suspect he may have included some of these among his cases of pericarditis.

3. Rilliet's observations refer to *children* only, in whom this complication is believed to be more frequent: his results also *approach* to those of Dr. Macleod for children.

4. Dr. W. Budd and myself alone give data which admit of a fair comparison, and (if I include my *slighter* cases, as he probably did) the results are very nearly the same, and certainly agree as closely as could be expected, when we consider the comparatively small number of cases from which they have been obtained.*

* In Dr. Latham's lectures, there are some important statements bearing upon the questions just discussed.

Out of 136 cases of acute rheumatism treated by Dr. Latham, the heart was affected in ninety, and exempt in forty-six.

The disease was seated in the

Endocardium alone in . . .	63
Pericardium in	7
Endocardium and pericardium in .	11
And the seat was doubtful in . .	9

It thus appears that pericarditis occurred twice in every fifteen cases of rheumatism; and, including all the slight as well as severe cases, I find the proportion to be identically the same in my cases of rheumatism.

Dr. Latham found signs of valvular disease in rather *more* than one-half of his cases of rheumatism, and I found the same signs in rather *less* than one-half of my cases.

The general result of the preceding investigation appears to me to be,—

1. That acute inflammation of the heart has occurred less frequently as a complication of rheuma-

The coincidence between these results of pure observation (as far as they have been given, and therefore as far as they are comparable) and those already cited in the text, proves, I think, the substantial accuracy of both. It is in the *interpretation* of the observations that so great a difference appears, and I cannot but believe that the remarks I have made upon the statements of the authorities I have quoted, are to a considerable extent, although not equally, applicable to those of Dr. Latham. Dr. Latham (page 145) has spoken of all his cases of valvular disease in rheumatism as examples of *recent* inflammation; but in a portion of them only has he given us any *proof* that the disease was recent. It appears by the facts stated in Dr. Latham's work, at page 233, that the murmur supervened after the patient's admission, in thirty-six cases; and by other facts given, at page 147, that the murmur *ceased* ultimately in seventeen cases. All the cases in which either of these circumstances was observed may be accounted cases of *recent* endocarditis; we are not informed, however, in how many instances the murmur was *both* observed to *commence* and to *cease* in the *same individual*. The number of cases *proved* to be recent is therefore uncertain, but must be between 53 (36+17) and 19 (36−17). Dr. Latham has shown, in various parts of his work, that he is perfectly acquainted with the distinctions between recent endocarditis and old valvular disease (vide p. 97 to 116). The remarks he has made upon this subject agree in substance with those which I have already made (vide note to p. 496). It may therefore be said, that he has kept these distinctions in view in making his observations, of which he has only given us the results. But I would urge that it would be very unsafe for us to assume this in the absence of any direct statement from the author to that effect, and still more in the face of

tism, in my experience, than it has been *believed* to occur in the experience of those writers whose opinions seem to have been most generally adopted by the profession.

some strong reasons for believing the contrary. One or two of these reasons I will state, for the question is a most important one.

1. The proportion of *all* cases of morbus cordis in acute rheumatism, found by Dr. Latham, as well as by the chief authorities I have referred to, is very nearly the same as that found by me. Now, I *know* that my cases comprise the examples both of old and recent disease, and I therefore *infer* from the agreement of that their cases comprise the same.

2. If *all* the cases mentioned by Dr. Latham were cases of *recent* inflammation, he has intentionally omitted all mention of cases of *old* valvular disease. Such an omission is in itself *improbable*; he nowhere tells us that he has made it, and he writes in such a manner as to leave the impression that he speaks of all cases of heart-disease, attended by murmurs, which he had observed.

3. Practical physicians are well acquainted with the frequent complication of pericarditis with pleurisy and pneumonia. Dr. Latham has adduced important evidence of this, and I have observed this complication in a large proportion of my cases. Dr. Latham has found the frequency of inflammation of the lungs to vary in different cases of rheumatism, as follows (p. 163):—

In cases of rheumatism without heart-affection, 1 in 9.

In cases of rheumatism with *endocarditis* alone, 1 in 9.

In cases of rheumatism with *pericarditis* alone, 1 in 2.

In cases of rheumatism with both endocarditis and pericarditis, 2 in 3.

Now, the remarkable *difference* in the proportion of cases of inflammation of the lungs, among the cases of pericarditis and those of endocarditis, and the equally remarkable coincidence of proportion among the cases of rheumatism, with endocarditis,

2. That the frequency of inflammation of the heart, even in my cases, has been such as abundantly to show the great influence of acute rheumatism in its production. To *prove* strictly the latter proposition, it would be necessary to show that inflammation, of the heart occurs less frequently in other acute diseases than in acute rheumatism. This I have not had the leisure as yet to accomplish; but with the caution now given, I think I may venture to make the required assumption.

Perhaps I may here record an opinion which I did not state in its proper place, because there are no published data to refer to in its support, but to which, on account of the scrupulous accuracy of the author, I attach all the importance that a verbal statement ought ever to receive. In 1841, M. Louis expressed to me his strong conviction that inflammation of the heart occurred much less fre-

and without any heart-affection, admit of a ready explanation on the supposition, the truth of which I am attempting to prove, that the cases called endocarditis, included also all cases of old valvular disease.

4. The great difference in the *mortality* of the two classes of disease seems to favour the same view. Out of eleven cases of endopericarditis, three deaths occurred, but not one death among the sixty-three cases of simple endocarditis. This comparison, I think, proves that endocarditis is less fatal than pericarditis, but I believe it makes the difference appear to be greater than it really is, and the supposition that many of the cases called endocarditis were cases of old valvular disease, affords a ready explanation of the apparently very great disparity in the mortality of the diseases compared.—(Vide also page 497, paragraph 3).

quently as a complication of rheumatism, than it was then the fashion in Paris to believe.

2.—*Frequency of Morbus Cordis in* CHRONIC *Rheumatism.*—Out of 109 cases of chronic rheumatism *taken indiscriminately*, and treated in the same period, and by the same physicians as the eighty-six cases of acute rheumatism referred to at page 481, I find,

20	are stated to have had some disease of the heart, chiefly valvular, and in *most* cases probably of old standing.
2	only had acute pericarditis, and even of these one was doubtful.
87	had no disease of the heart, or none is mentioned.
109	

If we compare these cases of chronic with the eighty-six cases of acute rheumatism, which were observed under the same circumstances we find,—

1. That the proportion of cases of disease of the heart, *chiefly* valvular, and believed to be *chiefly* old, is nearly the same in both classes, being about 22·09 per cent. in the cases of acute rheumatism, and about 18·34 per cent. in those of the chronic form.*

* *At first view*, this result may appear to be inconsistent with the facts given in the note to page 494, inasmuch as the proportion of cases of morbus cordis in chronic *rheumatism*, above given, seems to be smaller by one-half, than in *all kinds* of *diseases*, taken indis-

2. That there is however a small excess of the variety of cases of morbus cordis under consideration among the cases of acute rheumatism, which is probably due to a greater proportion of examples of *recent endocarditis* in this form of the disease.

3. That the *amount* of the excess just referred to is 3·75 per cent., and assuming it to depend wholly upon *recent* endocarditis, these figures will represent the *amount* of the excess in the proportion of cases of *recent* endocarditis in acute rheumatism *above* the proportion in chronic rheumatism.

The difference between the amount of the excess of valvular disease in rheumatismus acutus, over that in rheumatismus chronicus, (viz. 3·75, as above,) and the amount of the excess of valvular disease in rheumatismus acutus, over that in "all diseases," (viz. 10·270, vide note to p. 494,) is 6·520; and this figure may represent the excess of the amount of valvular disease in rheumatismus chronicus, over that in "all diseases;" and since, further, this excess is probably due to *recent* endocarditis, and also since

criminately, as given in the place referred to. The two series of cases, however, are not *strictly* comparable, because those spoken of at page 494, were all observed for the express purpose of ascertaining the proportion of cases in which there was any degree of morbus cordis; and, therefore, under circumstances leading to the record, if not to the observation, of slighter forms of cardiac disease in them, than in the 109 cases of chronic rheumatism recorded without any such view. Indeed, from the statements made, page 505, it will appear that there is a greater proportion of cases of valvular disease in chronic rheumatism than in the class

the class of all diseases here referred to probably (for reasons given in the note p. 494) includes hardly any cases of recent inflammation of the heart, the same figures will probably represent nearly the *absolute* amount per cent. of recent endocarditis in cases of rheumatismus chronicus.

It is necessary, however, to remember that the figure 10·270 above referred to, is calculated from observations made by myself alone; whereas, the figure 3·75 is calculated from observations made by three other physicians conjointly, and which yielded a smaller proportion of cases of valvular disease than did my observations. The difference, therefore, just arrived at, 6·520, representing the amount of recent endocarditis in chronic rheumatism, if erroneous, will probably be so by making the proportion of endocarditis in the last-mentioned disease appear too great.

4. That the proportion of cases of *ascertained acute* inflammation of the pericardium, (and pro-

of "all diseases." Hence, that which at first view appeared an inconsistency, affords, when more closely examined, an additional proof of the truth of the statements to which it appeared to be opposed.

The consistency of this explanation *at once* with the facts just referred to, and with those to which it has been before applied at pages 484 and 492, last paragraph, viewed in connection with the statement that the observations consist of several series made at different times and places, and by different persons, will afford to those who reflect upon the subject, a strong confirmation of the truth of the explanation.

bably also, as we have just seen, of the endocardium,) is much greater in the course of acute than in that of chronic rheumatism.*—(Vide note to p. 509.)

* From the facts which have been given in the text, various other questions appertaining to the absolute and relative frequency of pericarditis and of endocarditis, in different circumstances, may be solved numerically, and more or less conclusively in different instances. For the sake of those persons who may not find the inquiry too repulsive, the following examples of a few such questions are here given.

(1.) *Comparative frequency of acute pericarditis and of acute endocarditis in acute rheumatism.*

1. From the facts stated at page 483, we may calculate that in the cases of acute rheumatism treated by me, there occurred eight per cent. of cases of acute pericarditis (or, including two slight cases, 10·66 per cent.), and in the other cases treated in University College Hospital, the proportion of pericarditis was 5·813 per cent.

2. The frequency of endocarditis must be calculated by the more circuitous process already given, (p.494, note,) and the result cannot therefore be so *absolutely* depended upon. I am only able to give the proportion in the cases treated by myself, and, as we have seen, it is about 10·270 per cent. These calculations would show that acute pericarditis and acute endocarditis occur with equal frequency in cases of acute rheumatism.

A similar result may be obtained by a mode of calculation somewhat different from No. 2, and more strictly comparable with it: thus,—

3. The proportion of acute pericarditis in all kinds of diseases taken indiscriminately, I have found from numerous observations to be 1·25 per cent. The difference therefore between the proportion in acute rheumatism, and that in *all* diseases, is 9·41 per cent. Compare this with the corresponding difference already given above, (viz., 10·270, in paragraph No. 2,) and which repre-

3.—*Frequency of other internal inflammations than those of the heart in* ACUTE *rheumatism.*

In the forty-seven cases of acute or sub-acute

sents the amount of acute endocarditis in acute rheumatism, and the result is an excess of 0·860 per cent. of acute endocarditis above the proportion of cases of acute pericarditis in acute rheumatism.

4. Now the proportions obtained in paragraphs Nos. 2 and 3 might approximate still more closely if we could make a just allowance for the following difference in the character of the data upon which they are based. The class "all kinds of diseases," referred to in paragraph No. 2, are those described in the note to page 494; and being noted at the time of the patient's admission, they exclude all cases of endocarditis or pericarditis supervening after admission. But in the class "all kinds of diseases," referred to in paragraph No. 3, the observations were noted after the patients were discharged, and the class therefore *includes* all those cases of acute disease which supervened in the hospital. Be it remembered also, that in this class would occur many cases of acute inflammation of the heart, complicating rheumatism and Bright's disease.

The correction which these facts show to be necessary, would raise the figure 9·41 by very nearly the proportion of cases of acute pericarditis occurring *in the course of* all diseases indiscriminately, and would thereby bring it nearer (and perhaps very near) to the figure 10·270 obtained by the first calculation, as given in paragraph No. 2.

(2.) *Comparative frequency of acute pericarditis and of acute endocarditis in chronic rheumatism.*

From the figures given at page 504, it seems that pericarditis occurred in chronic rheumatism in about 1·834 per cent. of the cases. I have no data which enable me to calculate *directly* the proportion of cases of acute endocarditis in chronic rheumatism; (owing to difficulties in diagnosis referred to at page 496, note;)

rheumatism, mentioned at page 480, the following complications were observed:—

but a method of calculating it *indirectly*, and somewhat rudely, has been given at page 505. The proportion resulting from that calculation would be 6·520 per cent.; consequently, higher than that of pericarditis; but, for reasons given p. 506, this result can only be regarded as a rude approximation to the truth.

(3.) *Comparative frequency of acute pericarditis and of acute endocarditis in "all kinds of diseases."*

1. From observations before referred to, I find that *acute pericarditis* occurs in all diseases indiscriminately, in the proportion of 1·25 per cent.

2. I have no means of calculating the proportion of cases of *acute endocarditis*, in a SIMILAR series of cases of all kinds of disease.

3. The proportion of cases of acute endocarditis in *fatal* cases of *all* kinds may, however, be calculated by Tables I. and III., pp. 537 and 541. From these tables, it appears there were six cases of acute endocarditis in 192 fatal cases of all diseases, i. e. 3·13 per cent. The corresponding per centage of cases of *acute pericarditis*, deduced from the same facts, is 4·68.

(4.) *Comparative frequency of acute pericarditis in rheumatismus acutus, rheumatismus chronicus, and in "all diseases."*

Acute pericarditis was found—

1. In *acute* rheumatism, in 8 per cent. of the cases (or including two slight cases in 10·66 per cent.).

2. In chronic rheumatism in 1·834 per cent.

3. In "all diseases," in 1·25.

The results numbered 1 and 3 are calculated from the observations referred to in the note pp. 507 & 8, paragraphs 3 and 4, and observed by myself; that numbered 2 is calculated from the cases described p. 504, and treated by the other physicians to the hospital. If we make all the calculations from the cases treated by the latter physicians alone, we shall obtain results more strictly comparable; and, numbering them as above, they are—

1. *In the head.*

8 had *pain* in the head: and of these eight, one had also drowsiness and convulsions, four giddiness, and one delirium.

1 had *insensibility*. This symptom occurred early in the disease, and lasted for a short time only. In

8 the state of the head is not mentioned, and in

30 the head was free from disease, or there was only rheumatism of the scalp.

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No. 1.—5·81 per cent. (p. 480—1.)

No. 2.—1·834 per cent. (p. 504.)

No. 3.—0·374 per cent.

(5.) *Comparative frequency of acute endocarditis in rheumatismus acutus, rheumatismus chronicus, and in "all diseases."*

We have already seen that acute endocarditis occurs—

1. In *acute* rheumatism, in 10·270 per cent. of the cases (note to p. 494, also note to p. 507 & 8, paragraphs 3 and 4).

2. In chronic rheumatism, in less than 6·520, note to p. 508, paragraph 2.

3. I have no means of calculating the proportion of cases of *acute* endocarditis in "all diseases" (vide note to p. 509).

Pericarditis and endocarditis, then, appear to occur with almost equal frequency, whether they are observed in connection with—

1. Acute rheumatism (note to p. 507). Or, 2. Bright's disease (p. 537). Or, 3. All diseases (note to p. 551). In chronic rheumatism alone, a considerable disparity is found; but, as has been already stated, the results which relate to this form of the disease must be received with considerable caution.

With respect to the relative frequency of pericarditis and endocarditis, I have noticed the following opinions in the works of those writers whom I have had occasion to consult.

Dr. Watson observes, that "when pericarditis happens, in the course of an attack of rheumatism, so, also, to the best of my

One had also erysipelas of the face.

Only about one-fifth therefore had symptoms of any affection of the head, and not more than one in sixteen (if so many) had any inflammation within the head.

2. *In the lungs.*

11 (*about* one fourth) had *bronchitis*. This was generally trifling in amount, and in three it is stated to have been chronic, and to have existed previously.

2 (one in twenty-three and a half) had pleuritis. In one the left was the side affected; in the other, both sides; in the last, there were also tubercles. In

18 the lungs are not mentioned, and in

16 there were no symptoms of disease in the lungs.

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belief, in almost every instance does endocarditis." (Lectures, vol. ii., p. 263. London, 1843.)

Dr. C. J. B. Williams believes that rheumatism "still more frequently produces endocarditis," than pericarditis (quoted already, p. 476).

Dr. W. Budd found endocarditis to be *four* times as frequent as pericarditis, in acute rheumatism (vide ante, p. 487).

All these opinions, but especially the last, *appear* at first sight to be opposed to the results I have given above; but, in reality, the difference is only *apparent*; for, whilst my numbers refer to *acute endocarditis alone*, those of Dr. Budd, and probably, also, of the other writers in question, *actually*, although not professedly, comprise both *acute* endocarditis, and *old* valvular disease (vide p. 493, paragraph No. 2; vide, also, the opinion of Dr. Latham, quoted in the note to p. 500).

3. *In the abdomen.*

There was no example of recent inflammation in the abdomen.

In the eighty-six cases of rheumatism mentioned at page 481, the following were the complications noted:—

1. *In the head.*

2 had *insensibility.* One of these had undoubted *meningitis* with effusion of serum and blood, the other is the one referred to already in the forty-seven cases last mentioned.

4 had *pain* in the head. Of these, one had also drowsiness and convulsions, one confusion of intellect, and two giddiness.

2 had *giddiness* alone.

2 had *delirium* alone. In

3 there was no symptom of disease in the head, and in

75 the head is not mentioned.

2. *In the lungs.*

8 (about one-eleventh) had *bronchitis*, chiefly chronic.

1 had pleuritis. In

5 there was no disease, and in

73 the lungs are not mentioned.

3. *In the abdomen.*

2 had acute peritonitis.

1 had icterus.

1 had diarrhœa.

In the forty-two cases of rheumatism treated by

me, and mentioned at page 482, the following complications occurred :—

3 (1 in 14) had *pneumonia.*
5 (1 in 8) had *acute* bronchitis.
1 had pleuritis.
1 had erythema nodosum.
1 had roseola (gonorrheal ?).
1 had conjunctivitis (do. ?).

In the rest there occurred no inflammatory complication.

These results are such as are furnished by the cases I have examined, but I do not attach much importance to them. The proportion of the cases in which the state of each organ is not mentioned, is so high as to justify us in believing that many instances of internal inflammations complicating rheumatism must have been omitted. I do not think that these facts warrant us in drawing any very positive conclusions respecting the relative frequency of pericarditis and of other internal inflammations in acute rheumatism. As far as they go, however, they seem to show that pericarditis occurs more frequently in these circumstances than any other inflammation, except bronchitis. Inflammations of the lungs, pleura, and parts within the head, are perhaps the next in order, and those in the abdomen are the least frequent.*

* In the work already cited, Dr. Latham gives the following facts :—Of 136 cases of acute rheumatism, inflammation of the lungs was found in 24, viz., pneumonia in 18, pleurisy in 2, and severe bronchitis in 4. The proportion of the whole is about

4. *Frequency of other internal inflammations than those of the heart in* CHRONIC *rheumatism.*

In the 109 cases of chronic rheumatism referred to at page 504, the following complications were noted :—

1 in 5½. This agrees very closely with the proportion in my 42 cases referred to in the text, which was about 1 in 4½.

In a previous note I have given the result of Dr. Latham's observations upon the relative frequency of inflammation of the lungs, in cases of rheumatism, with and without accompanying inflammation of the heart. It may be interesting to record here an analysis of 20 cases of pericarditis which I made with the view of determining the proportion of cases in which pleurisy and pneumonia complicate the cardiac inflammation. This analysis was made above a year ago, and for that reason it does not admit of so strict a comparison with that of Dr. Latham, as if it had been made for this purpose.

Among the 20 cases of pericarditis, pleurisy was found in 13. In 6, on the left side, in 3 on the right side, and in 4, on both.

Among the same 20 cases, pneumonia was found in 7, it was double in 4, on the left side in 2, and on the right side in 1.

I found however a considerable difference in the frequency of pleurisy and pneumonia, according as the pericarditis belonged to one or the other of the two classes of cases already described (page 453).

Of the 20 cases of pericarditis, 11 were *rheumatic*, and 9 were what, to avoid circumlocution, I will call *renal*.

In 5 of the 11 rheumatic cases, there was also acute endocarditis, but in none of the renal cases was this complication found.

Among the 11 *rheumatic* cases, pleurisy was found in 5, and pneumonia in 5. In 4 of the last there was pleurisy as well.

Among the 9 *renal* cases, pleurisy was found in 8, and pneumonia in 2.

Pleurisy therefore was more frequent in the *renal* than in the rheumatic cases, whereas the contrary is true of pneumonia.

1. *In the head.*—One had *acute meningitis*, one *delirium*, three *headache*, and four *giddiness*. *Hemiplegia* also was noted in *five* cases, but I have omitted to state whether it was of older date than the rheumatism. In 100 the state of the head is not mentioned.

2. *In the lungs.*—Bronchitis was found in five cases, pneumonia in two, pleurisy in one, and the lungs are not mentioned in 105.

3. *In the abdomen.*—Hepatitis occurred in *one* case. There was no abdominal inflammation noted in the rest. The remarks made upon the value of the results last given are equally applicable to these. As far as the cases go, however, they seem to show,—

1. That acute inflammations of other organs, as well as of the heart, are less commonly observed in the course of chronic, than of acute rheumatism.

2. That in the course of *chronic* rheumatism, pneumonia, pleuritis and meningitis occur as often as pericarditis does.

5. *Of the circumstances which favour the occurrence of pericarditis in the course of rheumatism.*

1. *Metastasis.*

The examples of the connection of rheumatism and pericarditis, we have seen (p. 454) were fifteen in number. In *ten* of these the rheumatism continued without any abatement after the pericarditis had supervened ; in *one* the rheumatism ceased, but not suddenly, about the time the pericarditis appeared ; and in *another*, the rheumatism gradually subsided,

and had altogether ceased for three or four days before the pericarditis began. In *one* case no information on the point in question is given. In *two* cases the pericarditis *preceded* the rheumatism, in one by a few hours only, but in the other by some days. In the latter case the rheumatism was most *trifling* in degree, and *transient* in duration, whilst the pericarditis was most severe and fatal, and the case may *perhaps* be viewed, as already suggested, (p. 454, also p. 519-22,) as an example of rheumatic fever without arthritis. If this opinion be adopted in respect to this case, it may perhaps be extended also to another case in which no articular affection was observed, (and which therefore I have not here *counted* among the cases of rheumatism,) but in which there existed pericarditis and profuse perspirations, without any *other cause* of the internal inflammation being discovered.

The above facts *prove*, I think, that *metastasis* is not a *frequent* cause of the pericarditis which occurs in connection with rheumatism, since I have not met with a single example of its operation; but they do not prove that such a thing as metastasis *never* occurs. It *may* be true that if, in the height of acute rheumatism, anything occurred to cause the somewhat sudden disappearance of the articular affection, (without at the same time removing from the system the *presumed cause* of that affection,) the heart would be more likely to suffer than in those cases in which the articular affection gradually subsides. The only mode by which the question could

be determined, would be to compare an equal number of cases in which the rheumatism in its progress has followed each of these courses, and to ascertain in which class the heart has suffered most frequently. This is a test however not very easily applied on account of the infrequency of those cases of rheumatism in which all external inflammation abruptly disappears. Perhaps an approximation to the truth might be arrived at by comparing those cases in which the articular affection is *more fixed* with those in which it exhibits a greater tendency to *migrate* from one joint to another (vide p. 523). If we thus permit ourselves for a moment to *go beyond* the evidence of the facts before us, without however *contradicting* it, and to *speculate* upon the evidence with which facts, not yet observed, may furnish us, (a procedure not only consistent with the philosophy of Bacon, but forming an important part of it, and exemplified abundantly in the history of the physical sciences,) we may easily frame an hypothesis consistent at once with the facts I have adduced, and with the doctrine of metastasis. *Assuming* the cause of rheumatism to be a morbid matter in the blood, we can conceive (what seems accordant with facts) that this matter has a greater tendency to fix itself in the joints, and to cause inflammation there, than elsewhere; that it has however a certain tendency to become fixed in other parts (especially some internal organs); that *in general* it locates itself in these parts without, at the same time, leaving the joints (perhaps from an excessive formation of the morbid matter);

but that if it do leave the joints *suddenly* (and not in consequence of being gradually destroyed or expelled from the system), as from any external cause, it will be more likely, than in other circumstances, to fix itself in the other parts for which it has an affinity.

The testimony of most observers agrees with my own, in tending to show that metastasis is not the *ordinary* cause of the development of pericarditis in the course of rheumatism. Some writers indeed seem to think that metastasis *never* occurs, whilst others appear to me to attach an undue importance to it. Perhaps the speculations I have just hazarded may help us to reconcile whatever is contradictory in the statements of authors upon this subject.

Cases of rheumatism, in which the cardiac inflammation appears before the articular.—Various writers have observed cases in which inflammation of the heart began before the articular inflammation in rheumatism.

1835.—Dr. Watson * gives one case in which the heart suffered first, and states that he has seen others; he refers, also, to a case related by Dr. Duncan, of Edinburgh.

1839.—Dr. Hope † has observed the same fact, and refers to a second case, which had been mentioned to him.

1835.—One of the best-marked examples, how-

* Lond. Med. Gaz., vol. xvi.

† Treatise on the Heart. 3rd edition, p. 178-9.

ever, of the occurrence under consideration is related by M. Hache.* The patient was *first* attacked with pericarditis, a very intense acute rheumatism supervened on the twelfth, and ceased on the twenty-first day. Then pleurisy of the left side showed itself, which had ceased entirely on the seventeenth day. The affection of the pericardium, after having slowly decreased from the fifteenth to the thirty-second day, was aggravated on the thirty-sixth, and was not altogether cured until fifty days from its commencement.

1842.—A very unequivocal case of the same kind is related by Dr. Graves.† The patient was admitted into the hospital for trifling febrile symptoms. On the fifth day afterwards, symptoms of pericarditis appeared, and on the tenth day severe articular rheumatism supervened, ran its usual course, and lasted ten or twelve days. The symptoms of pericarditis ceased about two days before the rheumatism appeared, and the heart exhibited no sign of disease throughout the course of this affection.

Acute rheumatism and rheumatic pericarditis without any articular affection.—Two of my cases (p. 454) seem to render the opinion not improbable, that as, in rheumatic fever, the articular inflammation *ordinarily* occurs without pericarditis, so, in some rare instances, the pericarditis *may* occur without any articular inflammation, *the cases being in all other respects the same.* The cases given by M. Hache

* Archiv. Génér. de Méd., t. ix., p. 325.

† Clinical Medicine, p. 915.

and Dr. Graves may be considered to have been of this description, before the *ordinary* symptoms of rheumatism supervened. It seems to me probable that, in these cases, the morbid condition of the general system was essentially the same in *all periods* of their course, and was also essentially the same as that which exists in ordinary cases of acute rheumatism. In *one* stage of this diseased condition, inflammation of the *heart* appeared; in *another* stage, inflammation of the *joints*. It has been *proved* that either of these inflammations may appear before the other; also, that the *articular* inflammation *commonly* runs its course without being accompanied by that of the heart. There seems, therefore, to exist a strong *à priori* argument in favour of the occasional occurrence of inflammation of the heart in rheumatic fever, without inflammation of the joints. This opinion implies the possibility of rheumatic fever occurring without arthritis, and is supported by the facts which support the latter opinion.

The doctrine that rheumatic fever may occur "without rheumatism," is strongly and ably advocated by M. Chomel, who considers such cases to be analogous to those, admitted by various observers, of small-pox, measles, and scarlet fever without any eruption.*

* For a fuller discussion of the doctrine in question, I would beg to refer to some valuable and original remarks by M. Chomel, on the analogies of what he terms "*disseminated inflammations*," and on the differences between these and other inflammations. In

Dr. Graves has witnessed "several well-marked cases of individuals liable to rheumatic fever, and who had previously suffered from attacks of fever,

this class he places small-pox, measles, scarlet fever, typhus, urticaria, pemphigus, zona, varicella, aphthæ, furuncles, rheumatism, the various inflammations of plague and syphilis, and also the metastatic abscesses which follow wounds, operations, parturition, and small-pox. The chief points of agreement which he notices, among these affections, are:—

1. The *unity* of each affection, although represented by a *multiplicity* of *local* affections developed simultaneously or successively over large surfaces, or in organs distant from each other. 2. They cannot be artificially excited by the common causes of inflammation.—3. Antiphlogistic remedies have generally no influence upon the *duration* of the disease, and often but an equivocal one upon its *intensity*.—4. Each of them has in general only one kind of termination.—5. Some of them, as urticaria, have but a momentary duration; others, like rheumatism, persist for an almost indefinite period of time.—6. They are developed under the influence of specific causes.—7. They are *secondary* inflammations; by which M. Chomel understands, that the patients who suffer from them are the subjects of a "morbid condition," or "diathesis," of which these inflammations are only the consequence. Finally, M. Chomel inclines to the opinion that the morbid condition referred to, consists in some alteration of the animal fluids, and particularly of the blood. He cites some of the diseases of this class as examples of those in which there is "manifestly an infection of the liquids,"—e. g. small-pox, measles, scarlet fever, oriental plague, and contagious typhus. (Leçons de Clinique Médicale faites a l'Hôtel Dieu de Paris, &c., Fièvre Typhoïde. 1834, p. 529 to 539.)

The peculiarities, just enumerated, of the disseminated class of inflammations, are perhaps still better seen in the gonorrhœal than in the ordinary form of rheumatism. The frequent combination of conjunctivitis with the affection of the joints, and the fact that

with arthritis in the usual form, and subsequently, on exposure to cold, were seized with symptoms of pyrexia, which, in intensity, duration, and every other particular, were identical with their former fevers, save and except that from beginning to end not a single joint was inflamed."*

Dr. Todd believes the *rheumatic diathesis*, without an actual paroxysm of rheumatic fever, to be a fertile source of those cardiac diseases which are met with in early life.† His cases seem to me to prove that a *less amount* of rheumatism than is usually called *rheumatic fever* may be accompanied with inflammation of the endocardium or pericardium.

2. Form *of rheumatism.*—If we adopt the division of febrile rheumatism into a fibrous and a capsular variety, according to the characters of each laid down by Dr. Macleod, I find that all my cases of rheumatic pericarditis occurred in the fibrous form of the disease. This result accords with the experience of Dr. Macleod, who found that all the cases of heart affection witnessed by him, save two,

some patients can predict the occurrence of both as soon as they contract a gonorrhœa, seem to show that the local affections are *secondary*, in the sense of this term already defined. If this reasoning be admitted, we must also admit that gonorrhœa, like syphilis, although necessarily a purely local disease at first, soon leads to the development of important morbid changes in the general system.

* System of Clinical Medicine, p. 914.

† Practical Remarks on Gout, Rheumatic Fever, &c. London, 1843, p. 111.

arose from the fibrous or diffused acute rheumatism.*

In connection with this observation, it is not unimportant to remark that the inflammation generally attacks a smaller number of joints, and is much more obstinately fixed in them, in the capsular, than in the fibrous rheumatism. These peculiarities are especially seen, I think, in gonorrhœal rheumatism, which I should place in the capsular class,† and I do not remember to have seen a case of acute inflammation in any of the tissues of the heart, in this form of the disease. These *facts* agree with the *speculative* considerations already suggested in connection with the subject of metastasis (page 517). Considerable allowance, however, must be made for the *fact* (according to my observation) that the *fibrous* is much *more frequent* than the capsular kind of rheumatism. Of 387 cases of rheumatism also referred to by Dr. Macleod, there were, of the *fibrous* form 226, of the *capsular* 81, and of the muscular, 80.‡

3. *Intensity of the rheumatism.*—The rheumatism

* On Rheumatism, pp. 46 and 113.

† Dr. Todd objects to the division of rheumatism into the fibrous and capsular varieties, believing the latter to be *gout*, and not rheumatism. I have seen various cases of *gonorrhœal* rheumatism, however, which differ widely in their characters, both from chronic rheumatism and the fibrous form of the acute disease, and which agree in many important respects with Dr. Macleod's description of *capsular* rheumatism, but which, nevertheless, have no claim to be considered identical with gout.

‡ Op. cit., ch. xiv., p. 151.

was *very acute* in two cases only, somewhat less acute in two others, sub-acute in ten, and there was a mere trace of it in one.

The rheumatism could be said to be very acute, therefore, only in about one-fourth of the cases. In all these, the pericarditis was *very acute*, and affected the whole of the organ, and in two of them it proved fatal. In all the remaining cases the pericarditis was less intense, in two the result was fatal, but in *one* only from the pericarditis. In this one the rheumatism was most *trifling* and *transient*, and the inflammation of the pericardium was severe and general, but somewhat chronic in its duration. From these facts I am disposed to conclude,—

1. That the violence and fatality of rheumatic pericarditis are *generally* greater in the cases in which the accompanying rheumatism is *very* acute, than in those in which it is sub-acute.

2. Whether pericarditis be more *frequent* in the very severe, than in the less severe form of acute rheumatism, is uncertain. The facts just given, *as far as they go*, are opposed to such a view, three-fourths of the cases having occurred in sub-acute rheumatism. This result however *might* depend upon sub-acute rheumatism, being the more common form of the disease, *if such be the fact*. The question could only be determined by comparing an *equal* number of cases of rheumatism, of great and of moderate intensity, and by ascertaining in which class pericarditis was of more frequent occurrence.

On the question just referred to, we find that the

testimony of observers is conflicting, but the greater number appear to favour the opinion, that the more severe the rheumatism is, the more frequent will be the pericarditic complication. Bouillaud speaks very decidedly on this point, in a passage already quoted (page 485). Dr. Copland* and Dr. Macleod† entertain the same view. Dr. Latham and Dr. Watson are the only writers whom I remember, who express an opposite opinion: they say that pericarditis "is not more to be looked for when the disease (rheumatism) is severe, than when it is mild."‡

Dr. W. Budd § believes that "rheumatic inflammation of the heart is most common in severe cases, especially when there is much fever, and the parts affected are numerous." He adds, "it is our opinion that it is the fever chiefly which tends to extend the rheumatic inflammation over a great number of parts, and thereby increases the liability of the heart, in common with other parts, to become affected with rheumatic inflammation." In support of the last-mentioned opinion, Dr. Budd refers to the observations of Louis, which show that the number and extent of the secondary lesions in pneumonia, typhoid fever, and many other acute diseases, are proportioned to the degree of febrile movement.

If it should hereafter be proved to be a fact that rheumatic pericarditis is most frequent when the

* Medical Dictionary, vol. ii., p. 195.

† On Rheumatism, p. 45.

‡ London Medical Gazette, vol. xvi., p. 164.

§ Library of Medicine, vol. v., p. 199.

fever is highest, it will still not necessarily follow that the *fever* is the *cause* of this greater frequency. It seems to me fully as reasonable to regard the *severity* of the articular affection, the *great number* of joints implicated, the inflammations in the heart, and the high fever, as *independent*, but *associated* effects of one *common* cause. The fever in rheumatism is not *caused* by the articular inflammation, (although it is *possible* it may be increased by it,) for it begins before, and is not always proportioned to, the local affection, but is most probably due to some change in the composition of the blood; if so, a *higher* degree of fever *may* imply a *greater* amount of change in the blood, and upon the same cause may depend the greater *extent* and *severity* of the articular, and the greater *frequency* of the cardiac inflammation. This view would also explain what appears to have occurred in my cases, viz., that pericarditis, when it *does* supervene, is *more violent* in proportion as the rheumatism is more severe. The argument drawn from Louis' observations does not seem to me to be strictly applicable: for, 1. There is no reason to believe that the inflammation of the heart in acute rheumatism is a *secondary* affection in any sense in which the term is not equally true of the inflammation of the joints; and, 2. Louis' remark that secondary lesions are most common in the diseases referred to by him, when there is most fever, may be quite true without obliging us to ascribe these affections to *the fever*. In such cases, compared with slighter ones, the illness is of long

duration, and there is much prostration of strength; circumstances capable of favouring in various ways the development of secondary lesions—to say nothing of the conceivable peculiarities of the original cause of the fever itself.

4. *Stage of the rheumatism.*—The pericarditis came on in two cases before the arthritis; in two on the first day of the disease, in three on the third day, in one on the fourth day, in three on the eighth or ninth, in two on the eleventh, in one on the seventeenth day, and in one the day was uncertain. In fully one half the cases of rheumatic pericarditis, therefore, the affection of the heart appeared on or before the fourth day of the disease. With one exception, the pericarditis did not appear sooner in those cases in which it was very severe, than in those in which it was much less severe.

5. *Influence of repeated attacks of rheumatism.*—In eleven out of the fifteen cases, the pericarditis supervened in the course of the *first* attack of rheumatism, in three during the second attack, and in *one* during the *third*. The eleven cases referred to, comprise all the examples of the *most intense* form of pericarditis. Hence rheumatic pericarditis appears to be both more *frequent* and more *severe* in the first, than in subsequent attacks of rheumatism.

6. *Previous disease of the heart.*—Ten out of fifteen patients had no previous disease of the heart, (and among them occurred *all* the *most severe* cases of pericarditis,) the rest probably *all* had. Whe-

ther, however, pericarditis be more frequent in those cases *of rheumatism* in which the heart was previously diseased than in others, these facts do not enable me to determine. The fact that it occurred in most of the cases during the *first* attack of *rheumatism* is an *argument against* such a supposition, but is not *conclusive*, because very many cases of organic disease of the heart have arisen independently of rheumatism.

7. *Age of the subjects of rheumatism.*—Of the fifteen patients having rheumatic pericarditis, nine, or two-thirds, were twenty years old or under, *five* between twenty and twenty-six years, and the remaining one only was above forty years of age. Rheumatic pericarditis therefore is more frequent in young persons than in old ones; but we must remember that acute rheumatism is itself more frequent in the earlier periods of life. These facts then do not meet the real question, which is, whether, among patients suffering from acute rheumatism, pericarditis be relatively more frequent in the young than in the old.

Dr. Macleod gives some figures which seem to show "that the proportion of cases of rheumatic pericarditis is considerably greater in young subjects, than in those more advanced in years. Of those referred to in the preceding table of cases of acute rheumatism, but eight were under 15 years of age; and of these, four, or half, had pericarditis. Between 15 and 20 there were forty-eight patients, and of these, sixteen had pericarditis, or one third;

between 20 and 25 years of age there were forty-six patients; and of these, sixteen had pericarditis, being very nearly the same proportion as the preceding; between 25 and 30 there were forty-two, and they afforded only ten, or not quite one in four. After this the proportion of cases rapidly diminishes, for from 30 to 35 we have twenty-seven cases of rheumatic fever, giving us only three of pericarditis, or one in nine; and from 35 to 40, we have also twenty-seven cases of rheumatism, without one of pericarditis."*

8. *Sex.*—Of my 15 patients, *nine* were males, and *six* females; but then rheumatism is also more frequent among men than women.

Dr. Macleod's cases however would seem to show a greater tendency to pericarditis in *females* than in males suffering from rheumatism: for "of the total number of patients labouring under acute rheumatism, 137 were males, and 89 females; of the former 28, and of the latter 24, had symptoms of pericarditis; or, of the men, rather less than one in five, and of the women rather more than one in four."†

9. *Previous strength of the patient.*—Four patients were robust or vigorous up to the time of the illness under which they were suffering. Of the *eleven* remaining patients, one or two were previously not very well, and the rest were not naturally strong. I am not able to state what proportions of all the

* On Rheumatism, 1842, p. 156-7.

† Op. cit., p. 154.

patients suffering from acute rheumatism were previously robust or weakly.

10. *Influence of Venesection.*—Twelve of the patients had not been bled before the pericarditis appeared, and among these are found all the *worst* cases of pericarditis, and all the fatal cases save one.

Three patients were bled, and in the manner following:—

One to ʒxvi, five days before the pericarditis appeared.

One to ʒxii, three days before the pericarditis appeared.

One to ʒxvi, eleven days before the pericarditis appeared.

In none of the three did the rheumatism abate, after the venesection, before the pericarditis supervened.

As far as they go, then, these facts contradict the supposition that venesection in rheumatism favours the occurrence of pericarditis, but they do not *quite* meet the real question, viz., whether among the subjects of *acute* rheumatism, those who are bled, and especially those who are *largely* bled, suffer from pericarditis in greater proportion than those who are not bled.

Dr. Alison and M. Gendrin believe that large bleedings favour the production of pericarditis. Dr. Alison "has no difficulty in stating his conviction, that large and repeated bleedings in the beginning of rheumatism, increase the risk of this metas-

tasis;"* and again he says, "the acute rheumatism cannot, probably, be much shortened in its duration by antiphlogistic remedies; and if it were so shortened in external parts, we have good reason to think that the risk of affection of the heart would be greatly increased. For although the translation to the heart has been observed to take place under all modes of treatment of the disease, yet it has been seen to follow large bleeding, and immediately consequent recession of the inflammation on the extremities, often enough and quickly enough to justify much apprehension of such a result, when the quantity of blood taken is so great, as to produce a marked and immediate effect on the heart's actions."†

It *may* be true that large bleedings, *when they are immediately followed by recession of the inflammation on the extremities*, favour the occurrence of pericarditis in rheumatism. I am acquainted with no facts opposed to this view; but in a point of so much importance, it would be desirable to have an exact statement of the number and other circumstances of the cases in which such an occurrence has been observed. Cases of the kind referred to must be rare, and the opportunity of seeing even *one* of them has never happened to me.

M. Gendrin states that during several years he

* Cyclopædia of Practical Medicine. History of Medicine, p. 95.

† Outlines of Pathology, and Practice of Medicine. Edinburgh, 1844, p. 362.

combated acute rheumatism by blood-letting, repeated sometimes "avec peu de mesure." He then observed pericarditis sufficiently often; and he could even cite cases in which this affection had supervened in the highest degree of debility induced by bleeding. He refers to one case in which pericarditis appeared after the eleventh bleeding, and on the seventeenth day of an intense acute rheumatism, affecting a young man. Since he has renounced blood-letting, as the principal means in the treatment of acute rheumatism, he has but rarely seen pericarditis supervene; and when it supervenes, it does not ordinarily become severe. M. Gendrin quotes the results of Bouillaud's practice in support of his own. Every one knows that Bouillaud bleeds both largely and frequently in acute rheumatism, yet he finds that one-half of these patients present symptoms of endocarditis, or of pericarditis. (We have already seen, page 485, that the proportion Bouillaud now gives is much higher than this.) Gendrin states that this is rather below than above the proportion, in the cases of rheumatism treated by himself by blood-letting, but not in those treated by another method. "In twenty-nine cases of acute rheumatism, treated by nitrate of potass in large doses," this writer had "met with only three cases of pericarditis, and in only one of these was the inflammation found to have any severity, and to call for any special treatment."*

M. Gendrin's cases, however, seem to me to

* Leçons sur les Maladies du Cœur, p. 426-7.

prove no more than this, that large bleedings do not always *prevent* the occurrence of pericarditis. More exact numerical data would be required to show the *relative* frequency of pericarditis in rheumatism, treated by free bleedings, and by other methods (p. 490, note). Before we can estimate the value of the argument drawn from Bouillaud's practice, we must be *well assured* that pericarditis *does* occur more frequently in the cases of rheumatism observed in his practice, than in that of other physicians. Dr. Macleod has drawn an opposite conclusion from Bouillaud's statements; first, however, assuming that blood-letting has scarcely been employed in France in the treatment of rheumatism, and that it was among these cases (and not among *his own*, treated by large bleeding,) that Bouillaud observed inflammation of the heart to be so common.*

Conclusion.—Upon the whole, the circumstances which *seem* most to favour the occurrence of pericarditis, in the course of acute rheumatism, are :—

1. The *fibrous form* of the disease as distinguished from the capsular.
2. A *first* attack of rheumatism.
3. An early age in the sufferer from rheumatism.
4. A high degree of intensity of the rheumatism (?).
5. An impaired state of health, or a naturally delicate constitution of body in the subject attacked with rheumatism (?).
6. *Mode in which rheumatism produces pericarditis.*

* London Medical Gazette, 1837-8, vol. i., p. 151.

1. Dr. Elliotson ascribes the pericarditis to "*sympathy* with the fibrous membrane of joints attacked by rheumatism."*

2. Bouillaud thinks the cardiac affection "occurs in virtue of the tendency to *extension*, to *diffusion*, which characterizes rheumatic inflammation," and he also directs attention to the importance of remarking the similarity of organization between the pericardium and endocardium on the one hand, and the fibro-serous tissue, which "is the essential seat of acute articular rheumatism," on the other hand, in our endeavours to explain the connection under our consideration.†

3. Dr. Macleod thinks "rheumatic fever" (or the fibrous form of rheumatism) implicates the membranes of the heart "by *extension*," but that *capsular* rheumatism becomes transferred to the pleura, or the membranes of the brain, by *metastasis*.‡

Dr. Hope approaches nearer the truth when he says the inflammation extends to the internal fibrous tissues, as it extends from one joint to another, "by the affinity of tissue."§

Dr. W. Budd has more distinctly conceived and

* Lumleyan Lectures. London, 1830, p. 9.

† Traité Clin. des Malad. du Cœur, 1st edition, p. 474.

‡ On Rheumatism, p. 114.

§ This expression is taken by Dr. Hope from *Bichat*, and seems to me to afford a good example of one of those distant glimpses of yet undeveloped truths, which are caught only by the piercing eye of genius. The *affinity of tissue* has been ably illustrated in a valuable paper, by Dr. W. Budd, "On Diseases which affect cor-

enunciated the same proposition, in describing the inflammation of the heart, "as one of a series of local affections, which implicate identical tissues in various parts of the body."*

The same fundamental idea seems to have occupied the mind of each of the writers quoted, but it has been, by degrees, more and more distinctly defined and expressed.

Were I called upon to express my own views upon this question, I would adopt the following *hypothesis*, as consistent with all the facts with which I am acquainted. The cause of acute rheumatism is probably the presence of some morbid matter (or an *excess* of some natural ingredient) in the blood, which has an especial affinity for the fibrous and fibro-serous tissues of the body, and which by fixing itself in one or more of these, induces various local inflammations. The similarity of the structures implicated, is probably the reason why rheumatic pericarditis, or endocarditis, often occurs at the same time with, or succeeds to, rheumatic inflammation in the joints, just as rheumatic inflammation in *one* joint occurs with, or succeeds to, that in *another*. The heart is more frequently (?) and more *severely* affected in *severe* cases of acute rheumatism, for *the same* reason that *more joints* are affected, and more *severely* affected, and also that more fever is present in such cases; *which reason*

responding parts of the body in a symmetrical manner," in the 25th volume of the Transactions of this Society.

* Library of Medicine, vol. v., p. 195.

may not improbably be a greater *abundance* of the materies morbi in the blood.

This hypothesis is perfectly consistent with the supposed *occasional* occurrence of metastasis; and also with the fact that this is not the ordinary mode in which the heart becomes implicated.

Rheumatism, in the ordinary acceptation of the term, is not, therefore, properly *the cause* of rheumatic pericarditis, as is *demonstrated* by those cases in which the inflammation of the heart precedes that of the joints. We have the same *kind*, and even, more nearly than is commonly considered, the same *amount*, of evidence for affirming the pericarditis to be the cause of the articular inflammation, which we have for affirming the latter to be the cause of the former. The true view I believe to be that which regards both in the same light, viz., as independent consequences of that general morbid state which is present in acute rheumatism.

II. OF BRIGHT'S DISEASE OF THE KIDNEYS, AS A CAUSE OF PERICARDITIS; AND (INCIDENTALLY CONSIDERED) OF SOME OTHER INFLAMMATIONS.

1. ***Frequency of Bright's disease as an observed cause of pericarditis.***—We have already seen (p. 457) that out of 31 patients affected with acute pericarditis, *nine*, if not *eleven*, had Bright's disease, and that in none of these cases could any other cause for the pericarditis be discovered.

2. *Frequency of pericarditis, and also of some other internal inflammations, in cases of Bright's disease.*—In order to determine this question, I selected from the notes of my *post mortem* inspections, those of *fifty* cases in which the patients had either *died* of Bright's disease, or were found, after death, to have this disease in a somewhat advanced stage. On examining these cases, I found,—

TABLE I.

	Pericarditis in	Endocarditis in	Pleuritis in	Pneumonia in	Meningitis in	Cerebritis in	Peritonitis in
1. Actually existing or acute	5 (& 2 ?)* or 1 in 10	4, or 1 in 12	11 (& 3?) or 1 in 4½	12 (& 8?) or 1 in 4	3 (& 1?) or 1 in 16	8† or 1 in 6	5 (& 2?) or 1 in 10
2. The remains of previous	7, or 1 in 7‡	21 (& 3?) or 1 in 2 nearly	33, or 1 in 1½	1	6 (& 1?) or 1 in 8	...	13, or 1 in 4
3. Both, 1 and 2	1	3	7	...	...	...	2
4. Neither 1 nor 2	33	24	10	27	22	27	25
5. Not examined or not mentioned	4	1	...	2	17	15	7
	52	56	64	50	50	50	54

I have not *sought* for other inflammatory affections in these cases, but have noted a few which arrested

* The figures within brackets, and followed by a note of interrogation, indicate the number of cases in which the existence of the disease to which they refer was doubtful, and which are to be added to the well-ascertained cases.

† Five of these eight were cases of both softening and hæmorrhage, but in which the softening was judged to have preceded the hæmorrhage. I have stated in the text, that, besides the cases of cerebritis and of meningitis, there were two cases of apoplexy and one of hemiplegia, without any morbid appearances after death. I believe many such cases, without visible morbid appearances, are examples of Bright's disease, in which the symptoms in the head result from the circulation of poisoned blood.

‡ *Adhesions* of the pericardium are alone referred to.

my attention in passing. Thus there were two cases of apoplexy and *one* of hemiplegia in which no morbid appearance could be found in the brain or spinal cord; three cases of erysipelas, two of œdema glottidis, two of inflammation and ulceration of the mucous membrane of the ileum and colon, one of phlebitis, and one of eczema.

Of the above fifty patients, the ages of forty-nine have been recorded, and are here subjoined. There were—

Under 10 years	0
10 years and under 20 . . .	4
20 „ „ 30 . . .	7
30 „ „ 40 . . .	14
40 „ „ 50 . . .	7
50 „ „ 60 . . .	13
60 „ „ 70 . . .	2
70 „ „ 80 . . .	2
	49

The average age of each patient was very nearly forty-one years.

As the question under discussion is one of some importance, I have collected some similar facts from the publications of several writers of authority.

The following facts are taken and re-arranged from a table of the morbid appearances observed in 100 cases, in which Bright's disease formed a prominent feature, during life.* Out of these 100 cases there was found—

* The table in question is published by Dr. Bright, in the Guy's Hospital Reports, vol. i., 1836, p. 380.

TABLE II.

	Pericarditis in	Endocarditis in	Pleuritis in	Pneumonia in	Meningitis in	Cerebritis in	Peritonitis in
1. Actually existing or acute	8, or 1 in 12½	...	16, or 1 in 6	6, or 1 in 16½	...	8 } † 1 }	12 or 13, 1 in 8½
2. The remains of previous	6, or 1 in 16½	21, or 1 in 5	40, or 1 in 2½	5, or 1 in 20	13, or 1 in 7½	...	13, or 1 in 7½
3. Neither 1 nor 2	...	...	29				
4. Not mentioned	58*	8 ("Heart")	26*	12	...	41*	68*

The correspondence between the results of Dr. Bright and myself is sufficiently close to confirm the general accuracy of both. The difference in some instances may, perhaps, be explained by the larger proportion of Dr. Bright's cases, in which the state of the organs has not been mentioned, e. g. those in the table marked with an asterisk.

M. Becquerel ‡ has examined the morbid appearances in forty-five cases of Bright's disease, observed by himself, and in eighty-four collected from various authors. Out of these 129, he found—

Acute pericarditis in 4 cases, or 1 in 32 (1 in 45 of his own).
„ *pleuritis* in 19 „ 1 in 6 (7 in 45, or 1 in 6½ of his own).
„ *pneumonia* in 22 „ 1 in 5 about.
„ *meningitis* 1 in 34 (which 34 alone were examined).
„ *peritonitis* in 12 „ 1 in 11 about (6 in 45, or 1 in 7½ of his own).
Erysipelas in 5 „ 1 in 26.

In 33 *fatal* cases, taken from a total of 69 cases

† Eight cases of sanguineous effusion and one of softening of the brain.

‡ Séméiotique des Urines. Paris, 1841, pp. 492-6.

of Bright's disease, given by M. Malmsten, D. M. et Ch.* of Stockholm, the following, among other complications, are mentioned:—

Pericarditis acuta	in 3, or 1 in 11
Pleuritis	in 2, or 1 in 16½
Pneumonia	in 3, or 1 in 11
Meningitis	in 1
Erysipelas	in 3, or 1 in 11
Phlebitis	in 2, or 1 in 16½
No complication	in 8, or 1 in 4.

The *ages* of these 33 patients were—

10 to 20 years . .	4 cases
20 to 30 „ . .	14 „
30 to 40 „ . .	9 „
40 to 50 „ . .	6 „

The age has been recorded in 74 of Dr. Bright's cases, and was—

Under 30 years in 19 cases.
Above 30 years in 16 cases.
40 to 50 years in 21 cases.
Above 50 and chiefly under 55 in 13 cases.
Above 60 years in 4 cases.†

Fifty out of 74, therefore, sunk before the meridian of life (in the forty-fifth year and below).

Of Dr. Malmsten's patients, 27 in 33 died at or under 40 years of age, and *all* of them under 50.

3. *Frequency of pericarditis, and of some other inflammations, in patients without Bright's disease.*—For the purpose of comparing them with cases of

* Quoted in the Encyclographie des Sciences Médicales. Bruxelles. Août, 1843, p. 40.

† These numbers amount to 73 only, but they are copied accurately.

renal disease, I have selected from my *post mortem* inspections 142 cases in which the kidneys were either healthy or were not the seat of any readily appreciable disease. Among these cases I found—

TABLE III.

	Pericarditis in	Endocarditis in	Pleuritis in	Pneumonia in	Meningitis in	Cerebritis in	Peritonitis in
1. Actually existing or acute	4, or 1 in 35½	2, or 1 in 71	20, or 1 in 7	17 (& 9?) or about 1 in 8	6 (& 3?) or 1 in 23⅔	5* (& 2?) or 1 in 28⅖	14 (& 1?) or 1 in 10
2. The remains of previous	7, or 1 in 20†	60 (& 11?) or 1 in 2⅖ about	85, or 1 in 1¼	6 (& 4?) or 1 in 23⅔	8 (& 2?) or 1 in 17¾	...	13 (& 1?) or 1 in 11
3. Both 1 and 2	...	...	7	1	...	...	1
4. Neither 1 nor 2	126	61	43	106	73	83	114
5. Not examined or not mentioned	5	8	1	1	50	52	
	142	142	156	144	142	142	144

The other inflammations noted *incidentally* were less numerous in these cases than among those of Bright's disease.

Of the above 142 patients, the ages of 140 have been recorded, and are subjoined. There were—

Under 10 years	5
10 years and under 20 . . .	16
20 „ „ 30 . . .	25
30 „ „ 40 . . .	26
40 „ „ 50 . . .	37
50 „ „ 60 . . .	24
60 „ „ 70 . . .	6
70 „ „ 80 . . .	1
	140

* Two cases of softening, with hæmorrhage; one of softening alone; one of carious bone from injury.

† Cases of *adhesion* of the pericardium.

The average age of each patient was $36\frac{1}{2}$ years nearly.

A comparison of this table with the corresponding one of cases of Bright's disease given at page 537 (and I know of no such comparison made by any author) will aid us materially in estimating the tendency of Bright's disease to produce various internal inflammations, the two series of cases being alike in all respects but two, viz., the presence or absence of renal disease and the age of the patients. Taking the sum of the *seven* kinds of inflammation selected, we find (exclusive of the cases marked doubtful) :—

(1.) 50 cases of renal disease yielded 48 examples of *acute* inflammations,—i. e. 96 inflammations in every 100 patients; and that these all occurred among 30 out of the 50 patients,—i. e. 60 per cent. of the cases were complicated with internal inflammations, and the number of these inflammations averaged more than $1\frac{1}{2}$ to each patient affected. Also,

(2.) 50 cases of renal disease yielded 81 examples of *previous* inflammations,—i. e. 162 inflammations in every 100 patients ; and that these all occurred among 45 out of the 50 patients,—i. e. 90 per cent. of the cases were complicated with some previous inflammation, and the number of these inflammations averaged above $1\frac{3}{4}$ to each patient affected.

On the other hand we find :—

(1.) 142 cases *without* renal disease yielded 68 examples of acute inflammations,—i. e. 42 inflam-

mations in every 100 patients; and that these all occurred among 51 out of the 142 patients,—i. e. not quite 36 per cent. of the patients had some acute inflammation, and the number of these inflammations averaged $1\frac{1}{3}$ to each patient affected. Also,

(2.) 142 cases *without* renal disease yielded 179 examples of *previous* inflammations,—i. e. 126 inflammations in every 100 patients; and that these all occurred among 100 out of the 142 patients,—i. e. about $70\frac{1}{2}$ per cent. of the cases were complicated with some previous inflammation, and the number of these inflammations averaged nearly $1\frac{1}{2}$ to each patient affected.

Conclusions.

1. The proportional number of *acute* internal inflammations therefore in the series of cases with renal disease is *double* that in the series without renal disease (the numbers being respectively 96 and 42 per cent.): the proportion of *patients* likewise among whom these inflammations were distributed is greater in the former than in the latter series of cases (the numbers being respectively 60 and 36 per cent.): but the average number of inflammations occurring in *each patient affected*, is *nearly* the same in the two series, being however a little greater in the cases of renal disease.

This conclusion is *confirmed* by a comparison of the relative frequency of traces of *previous* inflammation in the series of cases with and without renal disease respectively: *for*,

2. The proportional number per cent. of previous inflammations in the cases of renal disease is only about one-third greater than in the cases without renal disease (the numbers being respectively 162 and 126 per cent.), and the proportion of *patients* among whom these previous inflammations were distributed is still *nearer* the same in the two series (the numbers being respectively 90 and 70·5 per cent.).

Now why do we not find the *same excess* in the number of *previous* inflammations which we find in the number of *recent* inflammations in the series of cases with renal disease, compared with the series without it? Only two explanations of the fact have occurred to me: 1. Assuming that in the cases of Bright's disease a great proportion of the examples of *previous* inflammation were *produced* by the disease of the kidneys; the influence of this cause might be *compensated* by a *more advanced age* in the patients not affected with renal disease (for it may be stated as an established fact, that the longer a patient lives, and the more time, therefore, that is allowed for the application of the various causes of inflammation, the greater will be the number of inflammations found, after death, to have been produced). On examining the *average* age of the patients in the two series of cases, however, I find it is *nearly* the same; but is *somewhat greater* in the cases *with* renal disease.* This explanation, there-

* Of the cases of Bright's disease, the ages were examined in 49, and the *average* for each patient was found to be 41 years

fore is inadmissible. 2. We may suppose that the *previous* inflammations were due to the *same general causes* in *both* series of cases, and that the *excess* of *recent* inflammations in the one class was due to Bright's disease, and was accordingly found *only* after the disease of the kidneys was proved to exist. This is the view which I am disposed to adopt, and it is further confirmed by the *slight excess* of *previous* inflammations in the series of cases of renal disease *coinciding* with a *similar slight excess* in the average age of the patients in that series, as compared with the other. Some additional facts which support this conclusion will be adduced hereafter.*—(Vide note to p. 549.)

nearly; of the other class of cases, 140 were examined, and the *average* age for each was found to be 36½ years.

* From the facts given in the text, some other questions connected with the absolute and relative frequency of various internal inflammations in Bright's disease, as compared with other diseases, may be elucidated. As these questions are only incidentally connected with the chief subjects of the paper, and as the discussion of them involves details which to some might be tedious, I have thought it best to insert it here.

1. *The relative frequency of various acute inflammations due to Bright's disease alone.*

(*a*.) The various *acute* inflammations observed after death in the series of cases of Bright's disease, were found in the following order of frequency.—(Vide Table I.)

TABLE IV.

1. Pneumonia (in 24 per cent.)	5. Pericarditis (in 10 per cent.)
2. Pleuritis (in 22 ,,)	6. Endocarditis (in 8 ,,)
3. Cerebritis (in 16 ,,)	7. Meningitis (in 6 ,,)
4. Peritonitis (in 10 ,,)	

This table does not, however, *necessarily* represent the tendency

4. *Comparative efficacy of rheumatismus acutus and of Bright's disease, considered as causes of pericarditis, and of some other inflammations.*

1. In examining the whole of the cases of acute

of BRIGHT'S disease to produce any one of the inflammations enumerated in it as compared with any other, *because* Bright's disease, considered as a cause of inflammation, is here added to, and mixed up with, *all other* causes.

(*b.*) We must, therefore, endeavour to eliminate the *other* causes referred to. Now the different *acute* inflammations observed after death in the series of cases *without* renal disease, were found in the following order of frequency.—(Vide Table III.)

TABLE V.

1. Pleuritis (in 14 per cent.)	5. Cerebritis (in 3·5 per cent.)
2. Pneumonia (in 12 ,,)	6. Pericarditis (in 2·81 ,,)
3. Peritonitis (in 10 ,,)	7. Endocarditis (in 1·40 ,,)
4. Meningitis (in 4·2 ,,)	

If, then, we subtract the per centage frequency of each inflammation, as given in Table V. (which represents the influence of *all* causes *but* renal disease) from that given in Table IV. (which represents the influence of *all* causes) the series of numbers thus obtained should represent the *relative* frequency of each inflammation *due to Bright's disease alone*, and, consequently, the *force* of the *tendency* of this disease to produce each of the inflammations in question respectively. The result of this calculation is subjoined.

TABLE VI.

The *excess* of the one per centage over the other per centage just described, is for

1. Cerebritis	12·5	5. Endocarditis	6·6
2. Pneumonia	12·0	6. Meningitis	1·8
3. Pleuritis	8·0	7. Peritonitis	0·1
4. Pericarditis	7·2		

2. A comparison of the numbers just given (in Table VI.) with

pericarditis observed, we have seen (p. 457) that nearly twice as many were connected with acute rheumatism as with Bright's disease. We cannot, however, infer from this circumstance *alone* that

those contained in Table V. will show the *relative* FREQUENCY of various inflammations due to Bright's disease ALONE, as compared with ALL OTHER causes of inflammation, and, consequently, the force of the tendency of Bright's disease, as compared with that of all other causes conjointly, to produce each inflammation respectively.

In this way we find that Bright's disease produces—

TABLE VII.

1. *Endocarditis* almost 5 times as often as *all other* causes put together.
2. Cerebritis fully $3\frac{1}{2}$ ditto.
3. Pericarditis ,, $2\frac{1}{2}$ ditto.
4. Pneumonia just as often as ditto.
5. Pleuritis fully $\frac{3}{4}$ times (8 to 14) ditto.
6. Meningitis less than $\frac{1}{3}$ times (1·8 to 4·2) ditto.
7. Peritonitis one hundredth part (0·1 to 10) ditto.

The order of frequency of the various diseases, as exhibited in Table VII., differs from that in Table VI., and the reason of this will become apparent, upon considering that Bright's disease may have a *strong* tendency to produce a given inflammation, which "ALL OTHER CAUSES" together, may either—

i. Have, also, a *strong* tendency to produce, as, e. g. in the case of pneumonia and pleurisy; or—

ii. Have only a *feeble* tendency to produce, as in the case of endocarditis.

So that the tendency of Bright's disease to produce *endocarditis*, is *relatively* greater than its tendency to produce *any other* inflammation, yet *absolutely* less than its tendency to produce *pneumonia*.

3. We see, then, that the *absolute tendency* of Bright's disease to produce *pericarditis* and *endocarditis* (the diseases whose causes

the tendency of rheumatism to produce pericarditis is greater than that of Bright's disease, *for* the fact can be equally well explained on the supposition

are, more especially, the objects of investigation in this communication), is less than its *absolute tendency* to produce some other diseases, as, e. g. pneumonia and pleuritis; whereas its *relative tendency*, as *compared* with the tendency of *all other* causes put together, to produce the two diseases in question, *exceeds* its *relative* tendency to produce any other disease except *cerebritis*.

4. *Cerebritis* appears to be the inflammation which Bright's disease has the greatest *absolute* tendency to produce, and, with *one* exception, the greatest *relative* tendency likewise; and meningitis and peritonitis are, of all the inflammations inquired into, those which Bright's disease has the *least* tendency, whether absolute or relative, to produce.

5. Since, according to Table VII., Bright's disease produces pericarditis and endocarditis, $2\frac{1}{2}$ and 5 times respectively, more frequently than *all other causes put together*, it would seem to follow clearly, that it is *the most influential of all the causes* of these inflammations, and, consequently, MORE INFLUENTIAL THAN ACUTE RHEUMATISM, which has hitherto been regarded not only as the chief, but by many *almost* as the exclusive cause of the diseases in question.

This conclusion may possibly prove to be correct, and might be received as unexceptionably true, but for *one* source of fallacy in the facts from which it is deduced, viz.: The *relative* frequency of Bright's disease, *and* of *all other causes* of these acute inflammations, has been inquired into in *fatal* cases *only*. Now, many examples of acute pericarditis occur from rheumatism, and probably, also, from other causes (e. g. pleuritis), in cases which do not prove fatal, and, therefore, have not been taken into our account. No doubt many cases of pericarditis and endocarditis which occur in *Bright's* disease, *also*, do not prove fatal; but the *extent* to which this occurs in the case of each of the causes referred to, and the consequent extent of the source of fallacy to be allowed for, is not easy to be estimated, and is one of the difficulties (hereafter referred to —p. 556) which arise in comparing an *acute*, and generally not

that the *tendency* in question is *equal* in the two diseases referred to, *if* it should appear that acute rheumatism is a disease of much more frequent oc-

fatal disease, with a *chronic* and generally fatal one.[1]—(Vide further, pp. 560 to 564.)

6. We have already seen reason to think (p. 545) that the *previous* inflammations, of which the traces were found after death, were *due*, in great measure, to the *same* causes in the series of cases *with*, as in that *without*, renal disease; and that these causes must, therefore, have been distinct from, and had probably operated anteriorly to, the existence of any disease of the kidneys.

This conclusion is confirmed by an inspection of the following Table (Table VIII.), in which it will be seen: 1. That the *order of frequency* of the several *previous* inflammations is almost identically the same in the two series of cases;[2] and 2. That the per centage amount of the frequency of each inflammation is, in the most important instances, nearly the same in each series. The slight excess in the inflammations, which is observed in the one series over the other, is found, however, as might be expected, on account of the greater average age of the subjects, on the side of the cases accompanied with renal disease.

Still further confirmation of the result under consideration is afforded by the fact that the order of frequency of the *previous*

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[1] I find, on examination, that of the 142 patients dying without renal disease, four had acute, and one chronic rheumatism in their *last illness*. Of the fifty patients with renal disease, not one had either acute or chronic rheumatism in their last illness.

[2] Two diseases, pneumonia and cerebritis, are exceptions to this statement, but there are obvious reasons for the difference observed. Pneumonia and cerebritis are noted as very *infrequent* among the *previous* inflammations; no doubt because the *former* disease, when it does not prove fatal, *commonly* leaves little or no anatomical evidence of its previous existence, and because the *latter* is *very generally* fatal in its acute stage.

currence (e. g. *twice* as frequent) than Bright's disease.

In relation to the present question, therefore, it

inflammations in *both* series of cases, and of the *recent* inflammations in the series *without* renal disease, is the *same*.[1]

TABLE VIII.[2]

Showing the order of frequency and the per centage amount of the frequency of the several kinds of *previous* inflammations of which the traces were found after death in the series of cases with and without renal disease respectively.

Cases *with* renal disease.			Cases *without* renal disease.		
1. Pleuritis	in 66	per cent. of cases examined.	1. Pleuritis	in 59·8	per cent. of cases examined.
2. Endocarditis	„ 42	„	2. Endocarditis	„ 42·25	„
3. Peritonitis	„ 26	„	3. Peritonitis	„ 9·15	„
4. Pericarditis	„ 14	„	4. Pericarditis	„ 4·93	„
5. Meningitis	„ 12	„	5. Meningitis	„ 5·63	„
6. Pneumonia	„ 2	„	6. Pneumonia	„ 4·22	„
7. Cerebritis	„ 0	„	7. Cerebritis	„ 0	„

7. If we add together all the examples of each of the seven kinds of inflammation selected which were found in the *two* series of cases, the *sums* obtained will represent the *absolute* and *relative* frequency, and the per centage amount of the frequency, of each inflammation, from whatever cause arising, as observed in the

[1] Three diseases are exceptions to this statement, but the explanation is obvious. Pneumonia and cerebritis, (for the reasons stated in Note [2], p. 549,) and endocarditis. The last disease appears very *frequent* among the *previous* inflammations, probably because, in the slighter forms of it which were noted, e. g. slight thickening of the valves and adjoining endocardium, it is of very frequent occurrence; and *yet* it appears very *infrequent* among the *recent* inflammations, partly because it is not in itself fatal in the slight forms referred to.

[2] Calculated from Tables I. and III.

becomes a matter of importance to ascertain the relative frequency, among *physicians'* cases, of acute

bodies of persons dying of all diseases indiscriminately. This has been done for the *recent* and *previous* inflammations separately in—

TABLE XI.[1]

Recent Inflammations.	Per cent.	*Previous* Inflammations.	Per cent.
1. Pneumonia	in 19·8	1. Pleuritis	in 61·4
2. Pleuritis	,, 16·14	2. Endocarditis	,, 42·2
3. Peritonitis	,, 9·9	3. Peritonitis	,, 13·54
4. Cerebritis	,, 6·77	4. Pericarditis	,, 7·3
5. Pericarditis	,, 4·7	5. Meningitis	,, 7·3
6. Meningitis	,, 4·7	6. Pneumonia	,, 3·64
7. Endocarditis	,, 3·12	7. Cerebritis	,, 0

It is necessary here to notice some statements which, without explanation, would be likely to appear contradictory to each other.

1. We have found (note to p. 507-11) that in acute rheumatism, acute pericarditis occurs quite as frequently as acute endocarditis.

2. We have just seen, likewise, that in all *fatal* cases, whether those of Bright's disease, or those free from renal disease, acute pericarditis is rather more common than acute endocarditis (note to p. 545, et seq.).

3. But by Table IX., just given, it seems that *old* endocarditis was found, in *all* fatal cases, six times as often as *old* pericarditis: the one being in the proportion of 42·2, and the other of 7·3 per cent. If *recent pericarditis*, therefore, be quite as frequent as *recent endocarditis*, why should *old* pericarditis be so much *less* frequent than *old* endocarditis?

The reason is, that the examples of *old endocarditis* referred to,

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[1] This table has been calculated directly from Tables I. and III., and not *in*directly from Tables IV. and V., which would make a slight difference in the figures.

rheumatism, and of Bright's disease. To do this accurately, however, it would be necessary to undertake a series of observations with this express object in view; for, as acute rheumatism is a disease which is peculiarly incapable of being overlooked (at least in its usual forms), and as Bright's disease is almost *equally likely to be* overlooked in a large number of cases (seeing that it is of very frequent occurrence without dropsy, and without any other symptom likely to attract the attention of any but those who

comprise the slightest forms of the disease, such as a trifling thickening of the valves, or of the endocardium lining the ventricle: whereas the examples of *old pericarditis* with which the comparison is made, include only cases of *adhesion* of the pericardium, and consequently the more severe forms of the disease.

The proper cases for comparison with the above-described cases of *old* endocarditis, would be *all* cases of old pericarditis; including, besides cases of adhesion, the *slightest* forms of the disease, such as white spots, and even less alterations of the pericardium.

Now the cases thus described I have found to occur in the bodies of 105 out of 355 patients dying of all diseases indiscriminately which were examined by myself, and in *all* of which the condition of the heart was noted, i. e. in 29·5 per cent.

Mr. Paget, who counted *slighter* traces of the disease than I did, found these trifling cases alone to amount to 40·9 per cent. in *all* his *post mortem* inspections (On White Spots on the Surface of the Heart, in vol. xxiii. of the Transactions of this Society, p. 30); and Mr. Sibson, noting perhaps still slighter cases, found the proportion still higher (Transactions of the Provincial Medical and Surgical Association, vol. xii., p. 523-4).

Thus compared, therefore, the cases of *old pericarditis* are found to be at least as frequent as the corresponding cases of endocarditis, and they *may* be more frequent, and the contradiction above adverted to ceases to exist.

are *specially seeking* for the disease), all observations made under other circumstances would be sure to represent the number of cases of rheumatism, relatively to those of Bright's disease, higher than the truth. For these reasons, the tables published by the Registrar-General, and which are so valuable for many purposes, are utterly useless for this: for the same reasons, also, in part, and partly because the diagnosis was recorded, in general, on the first examination of the patients, and, consequently, before the urine had been tested, the series of cases observed by myself, and already referred to (in the note to p. 494), represent Bright's disease to be very much less frequent than it really is. With these explanations, I shall give such facts as I have collected; believing, however, that they will all be found tainted with the same error, although in very various degrees.

Dr. Bright found, from an extensive trial among the patients of Guy's Hospital, indiscriminately, that one in eleven presented signs of an affection of the kidneys. On another occasion he found the proportion to be one in six.*

In the Second Report of the Committee of the Statistical Society of London, on Hospital Statistics,† we find it stated that in ten London hospitals there were, on January 9th, 1843, 2,582 patients, the

* Quoted by Dr. Christison, in Preface to Treatise on Granular Degeneration of the Kidneys.

† Journal of the Statistical Society of London, Sept. 1844, vol. vii., p. 215.

names of whose diseases were specified. I have deducted from these cases all those which I suppose would be treated by the *surgeons* to the hospitals, and which I have calculated to be 1,461. Among the 1,121 cases which remain, there were 193 of rheumatism, (the *form* is not distinguished,) and 35 of disease of the kidneys, which I suppose to be chiefly examples of Bright's disease, since the cases of *nephritis*, ischuria, and diabetes, are excluded. This calculation gives one case of Bright's disease for every thirty-two physicians' cases, and 5·5 cases of rheumatism (of *all* forms) for every case of Bright's disease.

In the statistical tables of the Royal Infirmary of Edinburgh, we find the following facts recorded :*—

1*st Report*.—Number of *medical* cases admitted, 4,768.

No. of cases of Bright's disease, 77, or 1 in 61·9 of the whole.

No. of cases of rheumatism, (form not distinguished,) exclusive of pleurodynia, lumbago, and sciatica . } 339, or 1 in 14, about.

i. e. 4·4 cases of rheumatism (acute and chronic) for every case of Bright's disease.

2*nd Report*.—Number of medical cases admitted, 2,290.

No. of cases of Bright's disease, 48, or 1 in 47·7 of the whole.

No. of cases of *acute* rheumatism, 63, or 1 in 36·35 of the whole.

i. e. 1·3 cases of *acute* rheumatism for every case of Bright's disease.

3*rd Report*.—Number of medical cases admitted, 2,121.

No. of cases of Bright's disease, 34, or 1 in 62·38.

No. of cases of *acute* rheumatism, 69, or 1 in 30·74.

i. e. 2 cases of acute rheumatism for every case of Bright's disease.

* Appendix to the Reports regarding the Royal Infirmary of Edinburgh, 1841, 1842, and 1843.

In 1,014 *medical* cases occurring in University College Hospital, (note to p. 494,) there were found 83 cases of acute or sub-acute rheumatism, i. e. 1 in every 12·22 cases. There were in addition, 6 cases of gonorrhœal, and 112 of chronic rheumatism.

Some years ago, at my request, the urine of fifty-five patients, taken indiscriminately, and under the care of Dr. Elliotson, in University College Hospital, was examined by Mr. Weston, one of the clinical clerks. In two of these cases, the urine was albuminous, and of low specific gravity, and there was anasarca, and in a third the urine was albuminous, but the specific gravity is not mentioned, and there was no anasarca. The proportion of cases of Bright's disease, therefore, did not exceed one in eighteen of the cases examined.

The following facts are taken from cases treated by myself in University College Hospital :—

1. Season of 1841-2. No. of cases treated, 176.

No. of cases of acute or sub-acute rheumatism . . . . . } 9, or 1 in 19·5.

No. of cases of chronic rheumatism 15.

No. of cases of Bright's disease . 7, or 1 in 25·14.

i.e. 1·14 cases of acute rheumatism for every case of Bright's disease.

2. Season of 1842-3. No. of cases treated, 199.

No. of cases of acute and sub-acute rheumatism . . . . . } 24, or 1 in 8·3.

No. of cases of chronic rheumatism, 12

No. of cases of Bright's disease, 11 *certain*, 4 uncertain (the latter without dropsy) . . . . . } 15, or 1 in 13·3.

i.e. 1·6 cases of acute rheumatism for every case of Bright's disease.

As far as these materials go, they tend to show that the frequency of acute rheumatism, as compared with that of Bright's disease, in hospital practice, varies between the ratios of one and two, to one.

2. A second method of investigating the relative tendency of acute rheumatism and of Bright's disease to produce pericarditis, consists in taking an equal number of cases of each of those two diseases, and determining in which of the two pericarditis supervenes most frequently. This method, however, is liable to the objection already referred to, (note to p. 548, paragraph 5,) that rheumatic fever is an *acute* disease, and is, therefore, seen in its *whole* course; whereas, Bright's disease is generally a *chronic* affection, and, as treated in hospitals, is seen only in one point of its course. For the reasons just stated, I have not entered into this inquiry.

3. I have adopted, in addition to the first one, a *third* method of seeking a solution of the same problem, viz., by comparing a series of cases of acute rheumatism with a series of cases of Bright's disease, in which the renal affection was either found after the patient's death to be in an advanced stage, or in which it had been the actual cause of death. In this way, we have the *whole course* of *each* disease before us, and the results obtained should not be very far from the truth, in *relation* to *actually existing*, or *acute* inflammation of the heart, or other parts. In considering the relative frequency of the remains of *previous* inflammation

in the two classes, however, we must remember,— 1. That the average age of the subjects of Bright's disease is greater than that of those who have acute rheumatism; and, 2. That the course of the former disease is of much longer duration than that of the latter. Hence there is a greater chance in Bright's disease, than in acute rheumatism, of *other* causes co-operating; and also a greater chance of various inflammations arising from the *longer duration* of the renal disease, *apart* from the *mere strength* of its *tendency* to produce inflammation. If the *efficacy* of Bright's disease and of acute rheumatism, in producing inflammation in any given organ, be *supposed equal, then,* if the former disease run a longer course than the latter, it should give rise to a greater number of inflammations; and, consequently, *traces* of the previous existence of these should be found in greater number after death. The circumstances described would not exercise a similar influence upon the proportion of cases of *existing* inflammation. I shall now state some facts bearing upon the question under discussion.

1. *Pericarditis.*

Of 75 cases* of acute rheumatism, (p. 483,) 8 were complicated with pericarditis acuta, or 1 in $9\frac{1}{2}$.

* I have taken, for comparison, the last series of cases of acute rheumatism examined, because among them the proportion of cases of acute pericarditis is highest, and perhaps nearest the truth. I

Of 50 cases of Bright's disease, (p. 537,) 5 were complicated with pericarditis acuta, or 1 in 10.

Of 100 cases of Bright's disease, (p. 539,) 8 were complicated with pericarditis acuta, or 1 in $12\frac{1}{2}$.

Of 33 cases of Bright's disease, (p. 539,) 3 were complicated with pericarditis acuta, or 1 in 11.

Again. Of 20 cases of adhesion of the pericardium, (p. 464,) 9 had Bright's disease (4 of which had *also had* acute rheumatism); 5 had *had* acute rheumatism, of which 4 are those just referred to, as having Bright's disease also; and the *fifth* had *some* renal disease.

Hence, 1. Bright's disease and acute rheumatism appear to have caused *acute* pericarditis in an equal proportion of cases; and,

2. Adhesions of the pericardium (and, therefore, *previous* attacks of pericarditis) have been produced much more frequently by Bright's disease, than by rheumatism.

These *results* of *experience* are quite in accordance with a different series of facts given at page 546-56, as well as with the *views* developed in pages 556-7, and they are supported in some measure by the facts exhibited in Table I., page 537, and showing that one in seven of the patients dying with advanced

have excluded two doubtful cases of pericarditis, both from the class of rheumatism and of Bright's disease.

renal disease, have old adhesions of the pericardium.*

2. *Endocarditis acuta and valvular disease.*

(*a.*) In consequence of the diagnostic difficulties already referred to, (page 496, note,) we have no *direct* means of determining the frequency of *acute* endocarditis in rheumatic fever: the question may, however, be solved indirectly, although with a greater liability to error. Adopting the result given page 494 (note), we have

Acute endocarditis in 7·809 per cent. of cases of acute rheumatism,

And in Table I. p. 537 we have

Acute endocarditis in 8 per cent. of cases of Bright's disease.

(*b.*) In relation to the same question, we may usefully compare the proportion of cases of *old*

* There is a source of fallacy in the statements in the text, the value of which it is necessary to consider.

I have found that *acute* pericarditis is between three and four times as frequent among *fatal* cases treated by *physicians* in an hospital, as among *all* cases indiscriminately. We ought, therefore, to find acute pericarditis more commonly in *fatal* cases of *Bright's* disease, than in *all* cases of rheumatism indiscriminately.

The force of this objection, if not altogether destroyed, is considerably weakened by the following considerations:—

1. Pericarditis ought to be more frequent in rheumatic fever, as an *acute febrile* disease, (and *apart* from any allowance for its *special tendency* to affect the heart,) than in all diseases *indiscriminately*.

2. Pericarditis is more frequent in *fatal* cases of Bright's disease, than in fatal cases *without* renal disease; being found in one out of every ten of the former, and only in one out of every thirty-five and a half of the latter class of cases.

valvular disease, and of *recent* endocarditis *taken together* in rheumatic fever, and in Bright's disease. We thus find that

In 75 cases of rheumatismus acutus there was valvular disease, *old* or *recent*, in 34, or nearly half (page 483).

In 50 cases of Bright's disease there was valvular disease, old or recent, in 25, or just half (page 537.)

The similarity of the results exhibited in paragraph (*b*), and also of those in paragraph (*a*), (due allowance being made for the way in which the latter were obtained,) with those arrived at in relation to acute pericarditis (page 558), is a strong confirmation of the substantial accuracy of both. We must, however, remember that as we had the advantage of a *post mortem* inspection in the cases of Bright's disease,—paragraph (*b*)—*slighter* cases of valvular disease may have been detected among these than among the cases of acute rheumatism.

4. It is necessary, in this place, to recal some other facts which relate to the question under discussion. We have seen in a previous part of this communication that

1. *Acute pericarditis* occurs
2. ,,
3. ,,
4. ,,

Again we have seen that,—

5. *Acute pericarditis* occurs in 10 per cent. of the fatal cases of Bright's disease (p. 537).
6. „ endocarditis „ 8 „ „ „ (p. 537).
7. „ pericarditis „ 4·7 „ of ALL fatal cases (renal included). (Table IX.)
8. „ endocarditis „ 3·12 „ „ (Table IX.)

Hence by the results marked No. 1 and 3, we see that,—

(*a.*) Acute rheumatism produces acute *pericarditis* more than six times as often as do "all diseases" indiscriminately (*including* therefore all cases of acute rheumatism and Bright's disease, whether fatal or not). This result *may* be equally applicable to *endocarditis*, but facts are wanting to determine the point.

And by Nos. 5 and 7, we see that,—

(*b.*) Bright's disease produces acute pericarditis and endocarditis more than twice as frequently as all diseases indiscriminately. (Note,—these cases being *fatal* ones *only*, will therefore include many examples of Bright's disease, and but *few* of acute rheumatism.)

The results marked (*a*) and (*b*) agree sufficiently well with those (p. 558) which affirm an *equal tendency* on the part of acute rheumatism, and of Bright's disease, to produce pericarditis and endocarditis; allowance being made for the greater frequency of rheumatism than of Bright's disease, and also for the *small* number of cases from which they have been deduced. They would coincide exactly

if acute rheumatism were *three times* as frequent as Bright's disease, instead of being (as far as the imperfect data for making the calculation permit us to determine) not more than twice as frequent.—(Vide p. 556.)

But it also appears from other facts, which I have collected, but cannot conveniently record in this place, that,—

9. Acute pericarditis occurs in 0·467 per cent of "all diseases," exclusive of *acute* rheumatism (but inclusive of Bright's disease).

(*c.*) Hence by the results,—

No. 1 and 9, rheumatismus acutus produces pericarditis seventeen times as often as *all other* diseases put together, and (allowing rheumatism to be twice as frequent as Bright's disease, and the latter affection to be the *only other* cause of pericarditis, which is *not* the fact, still) eight and a half times as often as Bright's disease. But this conclusion is inconsistent with that (p. 558) which affirms Bright's disease to have an *equal* tendency with acute rheumatism to produce pericarditis, and *also* with that (note to p. 548, paragraph 5) which affirms that Bright's disease produces pericarditis two and a half times as often as all other causes put together, and consequently oftener than rheumatism, as well as with the facts stated at page 457, which show, that among a given number of cases of pericarditis, eighteen occurred in connection with rheumatism, and nine with Bright's disease.

Explanation.—The following considerations will reconcile these contradictions :—

In the note to p. 548, acute rheumatism is compared with *fatal*, or very advanced cases of Bright's disease; but in conclusion (*c*), page 562, acute rheumatism is compared with *all* cases of Bright's disease, consequently with those in the *incipient* as well as in the *advanced* stages. It would seem therefore a *probable* inference (because one which would *reconcile* the conflicting statements just given) that Bright's disease has a much greater tendency to produce pericarditis (and the same is probably true also of endocarditis) when in its *advanced*, than when in its *earlier* stages.

The inference thus somewhat *circuitously* arrived at, will be more *satisfactorily* established, if it shall be found to be confirmed by a *direct* comparison of the relative frequency of pericarditis in a considerable number of cases of Bright's disease in different stages. Until this shall have been done, the following considerations may further be adduced in support of it.

1. The impurity of the blood, to which he internal inflammations occurring in Bright's disease have been generally ascribed, is greater in the advanced than in the earlier stages of the disease.*

* These remarks (and indeed all others in this communication) have respect to the *chronic* form only of Bright's disease. It would be interesting to compare the *acute* with the *chronic* form in reference to the various conclusions stated in the text. It would also be interesting to compare the *acute* form of Bright's disease

2. In the advanced disease, the general strength of the patient is much more reduced—a circumstance which, however operating, is generally admitted to create a great liability to the development of various internal inflammations.

The final conclusions which the preceding considerations suggest, seem to be,—

1. That acute rheumatism and Bright's disease, *in its advanced stages*, have an *equal* tendency to produce pericarditis and endocarditis.

2. That acute rheumatism has a greater tendency to produce pericarditis and endocarditis, than has Bright's disease *when in its earlier stages.*

It will be interesting to institute a similar inquiry into,—

The relative frequency of some other acute inflammations, besides those of the heart, in rheumatic fever, and in Bright's disease.

1. *Inflammations within the head.*

Out of 47 cases of acute rheumatism (p. 480), there were perhaps three of acute disease, or inflammation in the head, i. e. . . . . 1 in 16

„ 42 other cases of acute rheumatism (p. 482), not one case of inflammation in the head occurred. Including both series, the proportion is 1 in 30

„ 50 cases of Bright's disease (Table I. p. 537), meningitis or cerebritis occurred in eleven, or . . . . . 1 in $4\frac{1}{2}$

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with acute rheumatism, in respect to their tendency to induce pericarditis and other inflammations. The acute form of Bright's disease is however comparatively so rare, that I am not able to make this comparison from the cases collected by myself.

2. *Pleuritis.*

Out of 47 cases of acute rheumatism, pleuritis occurred in . . . . 2 } or 1 in 30
„ 42 other „ „ 1 }

„ 50 „ Bright's disease, pleuritis occurred in . . . . 11 } or 1 in 4½
besides three doubtful cases }

3. *Pneumonia.*

Out of 47 cases of acute rheumatism, acute pneumonia occurred in . . . 0 } or 1 in 30
„ 42 other „ „ 3 }

„ 50 cases of Bright's disease, acute pneumonia occurred in . . . 12 } or 1 in 4
besides eight doubtful }

4. *Peritonitis.*

Out of 47 cases of acute rheumatism } No case of peritonitis or other acute abdominal inflammation occurred.
„ 42 other „ }

„ 50 of Bright's disease, there occurred five cases of acute peritonitis (besides two doubtful ones) . . . . } or 1 in 10

See, also, the Tables at pages 539 and 540.

It would appear, therefore, that Bright's disease, *in its advanced form*, is, much more frequently than acute rheumatism, complicated with inflammation of the pleura, lungs, peritoneum, and parts within the head. It is also worthy of being remarked, that the relative frequency of each of the inflammations referred to, except peritonitis, in Bright's disease and in rheumatism, is nearly the same, being about as seven to one.

This conclusion, however, is liable to *two* very serious sources of fallacy, viz. :—

1. The cases of Bright's disease were *all* fatal ones; whilst the cases of *acute* rheumatism were taken indiscriminately, and would not include many fatal ones. In the former class, therefore, whatever inflammation existed would be *discovered*; but in the latter, many inflammations, even if sought for, might escape detection, owing to imperfections in the means of diagnosis.

2. In a large number of the cases of acute rheumatism, the organs referred to above were either not examined, or have not been mentioned: in some, no doubt, they were passed over in silence, because they were found to be healthy, but in many they were probably not examined at all, and in these some internal inflammations may have been *overlooked*, and their *proportion* consequently underrated.—(Vide p. 508 to 513.)

3. OF THE OCCURRENCE OF PERICARDITIS IN OTHER BLOOD-DISEASES.

1. From the preceding facts and observations, we are justified, I think, in stating, that in all the cases referred to, there were observed but *two generic* causes of pericarditis. 1. Some disease of the blood. In *one* case this disease was *cyanosis*; in all the rest, either acute rheumatism, or *some* renal disease, but especially Bright's disease.—2. Extension of inflammation from a neighbouring texture to the pericardium.

2. I have already stated, that I do not put forth these as the only causes of pericarditis; neither do I

believe them to be so. But the facts detailed oblige me to conclude, that they are by far the most frequent, and therefore the most important.

3. Seeing that *two* diseases of the blood have been found to occupy so prominent a place among the causes of pericarditis, we might expect to find the same inflammation occurring, with more or less frequency, in the course of other blood-diseases. This consideration suggests inquiries of considerable interest, and calculated, perhaps, to lead to important results; but the subject is one upon which I am not now prepared to enter. Cases are recorded, showing that pericarditis does occur in various blood-diseases, e. g. in scarlatina,* in erysipelas,† in small-pox,‡ and in typhus fever; § but *isolated* cases are of little value in the determination of such a question. What we want to know is, the number of times that pericarditis, and other internal inflammations, were found in a *series* of cases of any given disease, *taken indiscriminately*, and in all of which the heart and other organs were carefully examined. A simple glance at the results would, then, show us what diseases have a tendency to induce various inflammations, the relative strength

* Dr. Scott Alison, in London Med. Gaz., vol. i., New Series, p. 664.

† Mr. Adams, in Dublin Hospital Reports, vol. iv., p. 371.

‡ Andral, Clinique Médicale—Maladies de Poitrine—Péricardite, vii^e^ observation.

§ Dr. Latham, Lectures on Diseases of the Heart, p. 348. And Mr. Paget, Med. and Chirurg. Trans., vol. xxiii.

of this tendency in different instances, and whether particular diseases, or classes of disease, have an especial influence in producing inflammation in one organ or tissue, rather than in others.

OF THE IMPORTANCE OF THE CONSTITUTIONAL OR PREDISPOSING CAUSES OF INFLAMMATION.

The facts which have been detailed in this communication appear calculated to exhibit the great importance of the previous state of the health of patients among the causes of inflammation, as distinguished from any single *external* or *exciting* cause.

Nearly all the cases of pericarditis which I have witnessed, were developed independently of any external or exciting cause, in the ordinary sense of the term. In those cases which were connected with renal disease, the cause of the cardiac inflammation must have been slowly produced, and gradually increased, by the operation of circumstances which interfered with the due depuration of the blood.

In the remaining cases, the application of cold was sometimes the *immediate* cause of the *rheumatism*, but the pericarditis was almost always induced *secondarily* through the rheumatism, and not directly by the application of cold, either at the same time with the rheumatism, or after the appearance of this disease.

I may take this opportunity of stating, also, my entire concurrence in the opinion of those who believe, that in very many cases of acute rheumatism, no undue exposure to cold or wet, or to the influence

of any other (if there *be* any other) acknowledged exciting cause of the disease, can be ascertained to have preceded the attack, within such a period of time as could justify us in regarding the two events in the relation of cause and effect.

I have examined a great number of rheumatic patients, with a special view to this question, and I therefore feel some confidence in the accuracy of the opinion to which I have referred. In these cases, the *predisposing* causes were the only ones which could be supposed to exist; and, if we further call to mind the great number of cases in which a very decided exposure to cold and wet has not been followed by rheumatism, we shall be justified, I think, in concluding that, whilst in some cases the *predisposing* are the ONLY causes of the disease, they are, in *all ordinary* cases, the *most important* causes, and that, without their co-operation, the acknowledged exciting causes would, in ordinary circumstances, be inoperative.

It has appeared to me, that acute rheumatism is frequently induced by the slow and continued operation of various agencies, whose effects are rather cumulative than very obvious, at any single point of time during the period of their application. Such agencies are, for example, habitual exposure to moderate degrees of cold and damp, inadequate clothing, bad or insufficient food, intemperate habits, and anxiety of mind.

In the case of rheumatic fever, and of Bright's disease, the particular change in the system to

which the local inflammations are due, would appear to consist in a vitiation of the blood. To the same general cause, we must also refer the local inflammations which supervene in the progress of various other diseases, and for which no *exciting* cause can be found. Such are the inflammations which complicate the eruptive fevers, glanders, phlebitis, &c.

These remarks have been suggested by the cardiac inflammations, which occur in rheumatism and in Bright's disease; but they are obviously equally applicable to the other inflammations, which occur in the same diseases.

May not these facts likewise be adduced in support of the probable truth of the observations of those physicians who maintain that no *exciting* cause can be discovered in very many examples of *various internal inflammations*, and who, consequently, insist especially upon the importance of a careful investigation into the previous health and habits of the subjects of these diseases.

CASE OF

EXCISION OF THE UPPER END OF THE FEMUR,

IN AN EXAMPLE OF MORBUS COXARIUS.

By WILLIAM FERGUSSON, Esq.,

PROFESSOR OF SURGERY IN KING'S COLLEGE, LONDON.

Received June 4th,—Read June 24th, 1845.

JOHN CLARK, ætat. 14. Admitted into King's College Hospital 20th November 1844. Always enjoyed good health until January last, when he first experienced severe pain in the left groin, which continued unabated, notwithstanding the use of leeches, blisters, seton, and the warm bath. Between three and four months after being first taken ill, the pain became most harassing in the left knee. Early in October a large collection of matter formed over and behind the great trochanter, which burst spontaneously, and there has been continued discharge ever since. The condition of the limb is such as is usually noticed in the advanced stage of hip-disease. It appears considerably shorter than the sound one, and is much bent both at the knee and hip-joints. There is constant pain in the knee, but there is little in the hip unless the limb be moved, or the head of the femur forced against the pelvis.

After remaining in the hospital for more than three months, the condition of this patient was as follows:—The distortion of the limb had increased, and in consequence of lateral curvature in the lumbar region of the spine, with corresponding obliquity in the pelvis, as well as further flexion at the hip and knee, the shortening was more apparent than ever. The heel on the affected side was between four and five inches above the other. The shaft of the femur sloped obliquely downwards and inwards, and the knee rested on the inner side of the thigh of the sound limb; the head of the bone could be felt, through the soft parts lying on the dorsum ilii, and its identity could be more accurately ascertained by passing the finger into a large sinus which opened on the surface over and behind the trochanter major. The articular extremity was so isolated that the finger could be passed round it in all directions. There were several sinuses contiguous to the large one, but it could not be ascertained that any of them led to disease of bone, or communicated in any way with the part. There was a large circular sore which occupied the whole of the skin over the trochanter major, with profuse discharge of thin matter from the open surfaces. The patient made no complaint of pain unless the limb was moved, but seemed weaker and more dejected than when first admitted: he could lie with comparative comfort, only on his right side. Pulse varied from 100 to 110; appetite indifferent; tongue very red; cheeks frequently flushed

After remaining in the hospital for more than three months, the condition of this patient was as follows:—The distortion of the limb had increased, and in consequence of lateral curvature in the lumbar region of the spine, with corresponding obliquity in the pelvis, as well as further flexion at the hip and knee, the shortening was more apparent than ever. The heel on the affected side was between four and five inches above the other. The shaft of the femur sloped obliquely downwards and inwards, and the knee rested on the inner side of the thigh of the sound limb; the head of the bone could be felt, through the soft parts lying on the dorsum ilii, and its identity could be more accurately ascertained by passing the finger into a large sinus which opened on the surface over and behind the trochanter major. The articular extremity was so isolated that the finger could be passed round it in all directions. There were several small sinuses contiguous to the large one, but it could not be ascertained that any of them led to diseased bone, or communicated in any way with the pelvis. There was a large circular sore which occupied the whole of the skin over the trochanter major, and profuse discharge of thin matter from the open surfaces. The patient made no complaint of pain unless the limb was moved, but seemed weaker and more dejected than when first admitted: he could lie with comparative comfort, only on his right side. Pulse varied from 100 to 110; appetite indifferent; tongue very red; cheeks frequently flushed; had

profuse night sweats, and the feet and face were slightly œdematous.

When this case first came under my notice, I entertained the hope that a spontaneous cure might ensue, but after watching its progress very carefully, and at the same time adopting such measures as appeared most likely to favour this result, I became convinced that there was much greater probability that the boy would die, as the system appeared to be giving way under the influence of the local disease. From an early date I had formed the opinion that the circumstances were peculiarly eligible for excision of the head of the femur, and that opinion was strengthened subsequently, when I had placed the point of my finger on that part. At first I was reluctant to subject the patient to such a formidable ordeal, but latterly fancied that I should be justified in resorting to such a step. The head of the femur appeared to act as a foreign substance amongst the soft tissues of the hip;—there was no clear indication of serious disease in the cotyloid cavity, and it seemed as if the contiguous bones were healthy. The boy's lungs and other viscera appeared sound, and the head and upper portion of the bone seemed for the time the sole cause of his sufferings.

With the concurrence of my colleagues, Mr. Partridge and Dr. Todd, and having had also the advantage of the opinion of my friend Mr. Coulson, I resolved to have recourse to the operation in question; and performed it on the 1st March 1845. A longitudinal opening about six inches long was made

in the line of the femur, extending from over the head of the bone to a little below the trochanter major, and the tissues were separated from the shaft of the bone, a little below that process, so as to permit a curved needle to be used for the introduction of a chain saw. This latter step was attended with considerable difficulty owing to the depth and obliquity of the bone, and when accomplished proved of little value; for after several trials the instruments (which worked very indifferently in my hands) broke, and I was compelled to adopt another mode of procedure. With a sharp-pointed bistoury I separated all the soft parts from the neck of the bone and the trochanters, and then, by causing the knee to be moved across the opposite thigh, and using the femur as a lever, the head and portion of the bone thus isolated was so thrust out of the wound that I could with facility apply the ordinary saw for the requisite section. Not being satisfied with the condition of the interior of the bone at the surface exposed by the saw, I enlarged the opening, and removed about three quarters of an inch more, then closed the wound with a few points of interrupted suture, and covered it loosely with a pledget of lint. No vessel of sufficient magnitude to require a ligature was divided. The cotyloid cavity was filled by a fibro-gelatinous mass, similar to the lining of the sinus.

When the patient was placed in bed, a long splint was applied, with a view of keeping up gentle extension; and the limb, as far as the bent state of the knee would permit, was placed parallel with its

fellow. The apparatus was somewhat similar to Boyer's long splint, but was so constructed that its upper end could be temporarily removed for the purpose of dressing the wound, and the fulcrum for extension, instead of being on the injured side, as when fracture of the neck of the femur is treated in this way, was taken from the other side of the pelvis.

There was scarcely any shock succeeding to the operation, and the chief complaint was pain in the knee, which for some days after was more severe than at any previous period. The symptomatic fever was very slight, and disappeared entirely within the first ten days. Part of the wound united by the first intention, the rest soon discharged healthy pus, and speedily showed a disposition to heal. The health improved rapidly, the sweatings ceased, as also the pain in the knee, the appetite became natural, and the patient gained flesh. On the 8th of May, the limb having been for some weeks completely straightened, and the wound having almost closed, the splint was removed, and the patient permitted to move about the wards upon crutches. He has since continued to improve in strength; moves about without trouble or pain; and wears his clothes as if in perfect health.

The length of bone removed was four inches and a quarter, measured through the curve of the neck and shaft, and the limb is now about two inches and a half shorter than its fellow. The cartilage was almost entirely removed from the head of the

bone, and the surface was in a state of ulceration. The trochanter and rest of the shaft seemed in a healthy condition, with the exception that the cancellated tissue was loaded with a dark-coloured fluid of an oily consistence, and seemingly mingled with imperfectly-formed pus. I am now doubtful whether this state of the interior was a morbid condition sufficient to necessitate the removal of the additional portion of bone.

This case, so far as I can learn, is the second of the kind attended with a favourable result which has occurred in Britain, and I have thought that it might prove an interesting communication to this Society.

The first case occurred to Mr. Anthony White in the year 1818, in the Westminster Hospital, and the history of the present one (so far as it has gone) is in almost every respect analogous to Mr. White's. In that example the patient was enabled twelve months after the operation to walk about with great facility, with the aid of a high-heeled shoe, and to execute all the ordinary movements of the limb. Many years after, the person died of phthisis, and the parts have been preserved in the Museum of the College of Surgeons. The upper end of the remaining portion of the femur was connected to the innominatum, nearly in the site of the acetabulum, by a strong fibrous band which had permitted free movement, for the purposes of progression, and at the same time been sufficient to support the weight of the body. I am informed by Mr. Cæsar

and Mr. Charles Hawkins, that Sir Benjamin Brodie has also removed the head of the femur; and Dr. Hargrave of Dublin has kindly forwarded to me some particulars of a similar operation which was performed in Dublin by the late Mr. Hewson. In both these instances the patients died, but the circumstances were not so favourable as in Mr. White's case and my own, for the parties were adults; and in Sir B. Brodie's, the head of the bone was in the acetabulum at the time of the operation. This patient died within a few days after, apparently the direct effect of that proceeding. Mr. Hewson's died three months after, from profuse suppuration, and the cotyloid cavity after death was found to be carious.

So far as I have been able to learn, these are the only examples in which the operation has been performed by British surgeons. Bourgery gives the names of ten surgeons in other parts of the world who have also performed it: of these patients, one half seem to have died from the proceeding.

An opinion prevails, that the celebrated Mr. White of Manchester was the first to perform this operation, and it is thought by some that Mr. Park of Liverpool also performed it. I believe however that neither of these gentlemen operated on the living body. If they did, the cases were never published. Dr. Radcliffe Hall has had the kindness to make particular inquiries for me on this subject at Manchester. Mr. Wm. Wood, the oldest of the surgeons in that town, who was house-surgeon of the hospital

in Mr. White's day, and Messrs. Ainsworth and Thorpe, the present senior surgeons, are positive that Mr. White did not perform the operation on the living body, and my friend Dr. Inman of Liverpool is as certain that Mr. Park never did it.

The error regarding Mr. Park may have arisen in consequence of his reputation for operations on joints, and I have no doubt that confusion has resulted from the two Whites being concerned in the history of the proceeding in question. The truth is, that Mr. Charles White of Manchester first proposed this operation in 1769, and that Mr. Anthony White first performed it in 1818, in this country. The suggestion was made by Mr. C. White in his remarks on the case of excision of the head of the humerus,—an operation he was the first to perform; and his words are, "I have in a dead subject made an incision on the external side of the hip-joint, and continued it down below the great trochanter, when on cutting through the bursal ligaments, and bringing the knee inwards, the head of the os femoris hath been forced out of its socket and easily sawn off; and I have no doubt but this operation might be performed upon a living subject with great prospect of success."*

P.S.—3rd October, 1845. This boy now walks about on crutches, and, besides looking hale and plump, expresses himself as being in every way in

* Cases in Surgery, &c., by Charles White, Manchester, 1770, p. 66.

good health. The limb is quite straight, and about the same length as when he left the hospital a month ago, although, as he moves along, the heel seems to be nearer the ground, on which he now rests the fore-part of the foot. There is free movement both at the knee and hip, and already at the latter part he has considerable power in elevating the thigh by the action of the psoas and iliacus internus muscles. There is no pain or tenderness about the hip, and the two sinuses have all but closed. The discharge from them is very slight, and there is no indication of any further disease in the bones.

ON THE

MINUTE STRUCTURE OF THE LUNGS,

AND ON THE

FORMATION OF PULMONARY TUBERCLE.

By GEORGE RAINEY, Esq., M.R.C.S.,

MICROSCOPIST OF ST. THOMAS'S HOSPITAL.

Communicated by RICHARD D. GRAINGER, Esq.

Received February 11th,—Read March 25th, 1845.

The lungs are made up of bronchial tubes, bronchial intercellular passages, and air-cells.

On the bronchial tubes and intercellular passages.—The bronchial tubes commence at the bifurcation of the trachea. They are composed of cartilaginous rings and a proper membrane. They ramify in the substance of the lungs, their cartilaginous rings gradually disappearing; and in the human lung, having arrived within about one-eighth of an inch of its surface, the membrane also terminates, but somewhat abruptly,* after which the passages conducting the air continue in the same direction as the bronchial tubes, of which they are the continuation, but without having any perceptible mem-

* See Plate V., fig. 1.

branous lining; their parietes being formed merely by the air-cells between which they pass and by which they are surrounded.

The membrane of the bronchial tubes retains its fibrous character as far as its termination, the fibres being arranged longitudinally and circularly, and also its lining membrane. These are supplied by a distinct set of blood-vessels, which at the termination of the membrane anastomose with the vessels of the air-cells. The diameter of the ultimate bronchial tubes is from $\frac{1}{50}$ to $\frac{1}{30}$ of an inch. They communicate with but few air-cells, and at these communications their membranous lining is not continued into these cells, but, on the contrary, the vessels of the cells pass into the bronchial tubes, and ramify very superficially on their inner surface, probably to allow the blood within them to be acted upon by the inspired air.

The bronchial intercellular passages are at first of a circular form, and, like the bronchial tubes, do not communicate with many air-cells; but as they approach the surface of a lobule, the number keeps increasing, and at length these openings of communication are so numerous, and so near together, that the intercellular passage loses altogether its circular figure, and becomes reduced to an irregularly-shaped passage, running between the air-cells, and communicating with them in all directions; lastly, having arrived close to the surface of a lobule, it terminates in an air-cell, which is not dilated, as stated by Reisseissen, but has about the same

diameter as the passage of which it is the continuation.* The epithelium (ciliated) which is peculiar to the air-passage, lines the bronchial tubes as far as the termination of the bronchial membrane, but it does not appear to extend beyond the membrane into the bronchial intercellular passages, or into the air-cells. The smallest tube in which I have seen the ciliated epithelium, had a diameter of about $\frac{1}{80}$th of an inch, but I have never found it in a tube where the bronchial membrane was not present also. As the lung near its surface is made up entirely of air-cells and intercellular passages, the contents of these can easily be examined, without there being any admixture of the epithelial lining of the bronchial tubes, care being taken only to select for examination those portions of the lung which lie immediately beneath the pleura. I have repeatedly examined these parts of the lung, but have not found this kind of epithelium. In the healthy lung, black pulmonary matter is present in greater or less abundance in the air-cells, as well as in the cellular tissue, distending the interlobular fissures and adhering to the external surface of the blood-vessels; and, in the diseased lung, granular matter may sometimes be found in the intercellular passages and air-cells, but it has not the character of ciliated epithelium.

In the uninjected lung, the remains of nucleated cells in the walls of the capillaries have a sufficient

* See Plate V., fig. 2.

resemblance to epithelium to be easily mistaken for it. Mr. Addison seems, from the following observation, to have fallen into this error:—"They (the air-cells) possess an epithelium, in the form of round nucleated scales, and from one to fifteen or more nuclei may be counted in a single scale." Mr. Addison, however, adds,—"But I have never satisfied myself that they possess the ciliated cylinder epithelium, so abundant in the trachea and bronchi."*

From what has been stated, it appears that the upper part only of the air-passages is lined by mucous membrane, and it will be shown hereafter that those parts in which the aeration of the blood more particularly takes place, are lined only by a very thin fibrous membrane. These facts seem to agree with the phenomena presented by acute inflammation of these structures, inflammation of the bronchial membrane (bronchitis) being attended by the symptoms peculiar to inflammations of other mucous membranes, and inflammation of the membrane lining the air-cells (pneumonia) being accompanied by deposition of fibrine, as in inflammation of the common fibro-cellular tissue.

The existence of a difference in structure between the membrane lining the bronchi and that lining the air-cells was long ago inferred, and insisted upon, by Dr. Addison and others, from pathological considerations; but, I believe, the fact has not been *demonstrated.*

* Philosophical Transactions, 1842, Part II., p. 162.

On the air-cells.—The air-cells are small, irregularly-shapen, and, most frequently, four-sided cavities, varying in size in different parts of the same lung, those being the smallest, as well as the most vascular, which are situated nearest its centre, whilst their size gradually increases, and their vascularity diminishes, as they extend into the more remote parts. This difference in size and vascularity of the air-cells is probably for the purpose of adapting the quantity of blood requiring to be decarbonized, to the deteriorated condition of the air in the differently situated cells. The renovation of the contents of the more superficial cells taking place more slowly than that of the contents of the more central ones—it being in the former effected by the passage of the air from the terminal into the more central cells (in consequence of the law of diffusion of gases), and in the latter, chiefly by the mechanical dilatation and contraction of the thorax, —it is necessary that there should be a proportionally small quantity of blood circulating through the former, than through the latter.

The air-cells which are situated close to the bronchial tubes, or intercellular passages, open into them by large circular apertures, whilst those which are placed further from these passages communicate with them through the medium of other cells. These communications of one cell with another are of the same shape and size as those which exist between the first set of cells and the bronchial tubes; and they can be seen very distinctly by looking into

the air-cells from the intercellular passages, and regulating the distance of the object-glass according to their different depths. As these openings are not necessarily in a straight line, the exact quantity of cells which communicate cannot in this manner be determined, but the number will depend upon the distance which intervenes between any given part of a bronchial passage and the surface of a lobule; so that, when a bronchial passage arrives nearest the surface, it will be separated from it only by a terminal cell, as before observed.

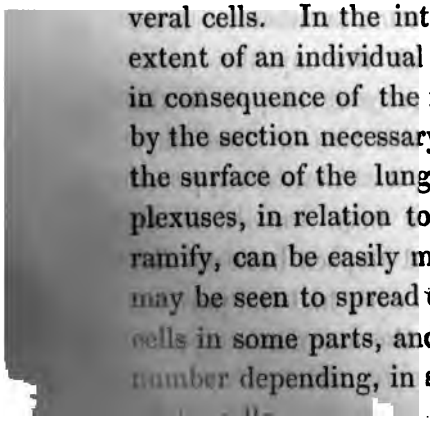
Besides these intervening air-cells, there are others which fill up the angle formed by the bifurcation of the intercellular passage, and which thus appear to form a cellular communication between them.

It is very easy, in an injected preparation, to see the communication between two, or sometimes three cells; but, to determine with exactness the number of cells which communicate in succession, is probably impossible, in consequence of the section which is favourable for displaying the opening of one, being unfavourable for displaying that of the other; but, that the communications between the cells of the same lobule are very free, is obvious from the fact, that, if injection be thrown into a small bronchial tube it will distend all the cells of that lobule, while none of the injection will pass into the adjoining lobules. In the mammal, the walls [illegible] separated [illegible] of vessels [illegible]

sacculi of the lung of the reptile, which may be considered to correspond to the air-cells in the lung of the mammal, are separated by a plexus folded on itself, and, therefore, consisting of two layers of vessels, a character by which the lung of the reptile differs from that of the mammal.

The plexuses in the lungs of the reptile and mammal consist alike of a very dense net-work of capillaries, into which terminal branches of the pulmonary arteries terminate, and from which the radicles of the pulmonary veins take their origin.

In the latter—the mammal—the number of capillary plexuses is not, as some have supposed, the same as that of the air-cells, that is to say, a terminal artery does not divide into a plexus at any particular part of a cell, its branches uniting for the commencement of a vein on the opposite part. On the contrary, one plexus passes between, and supplies several cells. In the interior of the lung, the exact extent of an individual plexus cannot be determined, in consequence of the removal of some part of it by the section necessary for its exhibition. But, on the surface of the lung where the extent of these plexuses, in relation to the cells over which they ramify, can be easily made out, an individual plexus may be seen to spread over an area of ten or twelve cells in some parts, and fewer in others, the exact number depending, in some measure, upon the size



has been removed, the terved descending from the

interior of the lung between the cells to the surface, where they send off their branches in all directions, some anastomosing with the adjacent branches of the pulmonary artery, and others terminating in the radicles of the pulmonary veins.

On that side of a cell in which is situated the opening of communication with an adjoining one, the capillaries anastomose all around this opening, so as to appear in the injected preparation, by reflected light, to form its immediate boundary; but by transmitted light, this opening will be seen to be formed by circular threads of the lining membrane of the air-cells, which extend beyond the circle of vessels.* To these communications, Mr. Addison has given the name of "lobular passages," under the idea that no air-cells exist in the fœtal lung, but that they are formed after birth by the mechanical dilatation of the bronchi.

The incorrectness of this notion is obvious from this fact, that the air-cells, with the capillary plexuses between them, are perfectly developed in the lungs of animals which have died two or three days after birth; for, in such cases, it cannot be supposed that these vessels could have been formed in so short a time as that which had elapsed between the birth and death of the animal, even had the parietes of the bronchi been dilated in the manner supposed by Mr. Addison.

With a view of settling this question, at the suggestion of Mr. Grainger, I injected the lungs of va-

* See Plate V., fig. 3.

rious fœtal animals, which had never breathed, and found, upon examining them with the microscope, that the air-cells were developed proportionally with the other parts of the lungs.

In the very young fœtus, the septa between the air-cells consists almost entirely of minute cellules or granules, and a small quantity of fibrous tissue, with scarcely any blood-vessels, so that the injection, which had been thrown into the pulmonary artery and extended into these vessels, became extravasated among the granules situated between the air-cells.

As the age of the fœtus advances, the granular matter diminishes, and the capillaries increase, so that at birth, the same arrangement of the air-cells and the other parts of the lungs exists as in after-life.

It has already been stated that the capillaries of the lungs are situated in a fold of membrane.* Proceeding with a description of this membrane, from the peripheral to the central parts of these organs, it may be described as lining, first the air-cells, which are next to the surface of a lobule, that is, those which are situated immediately beneath the pleura, and those which bound the interlobular fissures, being separated merely by the capillaries from the pleura in the one situation, and by the capillaries from the interlobular areolar tissue in the other.

From these cells it passes into the next set, enclos-

* See Plate V., fig. 3.

ing the capillaries, which are situated between them, and forming the immediate boundary of the opening by which they communicate: thence extending from one cell to another, it arrives at the intercellular passages, and, at the termination of the bronchial tubes, becomes confounded with the bronchial membrane.

This membrane is so thin and transparent that it can be seen only by transmitted light; it is distinctly fibrous; its fibres are strongest and best marked the nearer they are situated to the openings of communication between the air-cells, and some circular fibres extending a little beyond the vascular anastomoses which surround those openings, form their well-defined border.* This membrane besides lining the air-cells, and supporting the capillary plexus, will serve especially by means of its circular fibres to keep the openings into the cells patulous. Its structure has no resemblance whatever to muscular fibre, either of the striped or unstriped kind.

On the formation of pulmonary tubercle.—Having now considered the anatomy of the lungs, I will proceed to speak of the formation of pulmonary tubercle, and to show the manner in which the tuberculous deposit takes place, and extends itself by the destruction of the various parts of the lungs.

In order to determine with accuracy the precise situation of tuberculous matter, and to observe the manner in which the lungs become progressively destroyed by its presence, it is advisable to examine

* See Plate V., fig. 3.

tuberculous lungs which have been successfully injected.*

In such preparations, when viewed by reflected light, the pale colour of tuberculous matter contrasts so strikingly with the red colour of the injection in the capillaries of the walls of the cells, that there is no difficulty in discerning its exact form and limit, even in quantities so small as to fill only one, or even a small portion of a cell.

By this mode of examination, it will be clearly seen that the tuberculous matter is poured from the free surface of the pulmonary membrane into the interior of the air-cells. These becoming distended, and the septa between the contiguous cells being at first compressed, and their vessels afterwards obliterated, the supply of blood to the diseased part is cut off, and a tubercle formed, corresponding in size to the number of distended cells.

The manner in which tubercles extend themselves to the parts adjacent is best seen by observing the progress which had been made by the deposition in the air-cells situated in their vicinity. Here some of these cells will be seen to contain only a small quantity of deposit, others to be completely distended with it, though their vascular walls remain entire. Between other cells containing tuberculous matter, these vessels will be seen to have become partially obliterated; and lastly, in the septa, between those cells where the accumulation has amounted to such a quantity as, generally, to com-

* See Plate V., fig. 4.

press the capillaries, and probably cause their absorption, no vessels are left, and nothing exists but the pulmonary membrane, which remains mixed with the tubercular matter, and which (the tubercle having been broken up) is in a state to be ejected from the lungs in the expectoration, in which it can be detected by the microscope.

An imperfect examination of the vascular connection of a tubercle with the surrounding air-cells, in the injected lung, might give the idea of vessels passing into it from the adjacent unaffected parts, and thus lead to the supposition that it is vascular, —an idea which has long been prevalent among pathologists. A careful examination, however, clearly shows that these vessels are merely portions of plexuses which have not yet become absorbed. In most cases, these vessels will appear as arcs of circles of greater or less extent, of the same radius as the plexuses between the air-cells, of which they are, obviously, the remains.

The deposit of tuberculous matter takes place in the bronchial intercellular passages at the same time and in the same way as in the air-cells; and their walls,—which are in reality the air-cells between which they pass,—disappear in the same manner as those which separate one cell from another; and thus these passages being occupied by tuberculous matter, contribute to form a part of the tubercle. The smaller bronchial tubes also becoming distended with tuberculous matter, are involved in the general mass.

It will be obvious from what has preceded, that as a tubercle increases in size, the central parts of it will become further and further removed from those vessels, by which the tuberculous matter was in the first instance deposited, and afterwards maintained in a state of vitality, and consequently these parts will have the greatest tendency to lose their cell or vegetable life, and become softened. According to this explanation, this process ought always to begin in the centre of a tubercle, and I believe this is generally the case. But it must be recollected that the geometrical centre of the mass is not necessarily the point furthest removed from the source of circulation; the point may even be on the side of the tubercle, provided it be the remote cells of a lobule which are occupied by the tuberculous matter, namely, those bounded by an interlobular fissure: in such a case, this part of the tubercle might be further removed from the source of circulation, than its exact centre.

Some pathologists contend that tubercle is the result of inflammation. Without entering at length upon the consideration of this question, which is one *de verbo* and not *de re*, I may state that the perfectly natural appearance of the vessels close to a tubercle, and even of the cells containing a small quantity of tubercular matter, not sufficient to have impeded their circulation in the capillaries during life, when compared with the tortuous and unequally dilated state of vessels going to air-cells filled with fibrine, in consequence of inflammation, are patho-

logical considerations in favour of the non-inflammatory nature of phthisis. These facts go also to show that the obliteration of the capillaries between the air-cells is, in phthisis, produced by some force which has exerted upon them a slow and very gradual compression, such as can only be conceived to have been produced by the accumulation of tuberculous matter, in contiguous cells, pressing upon the intervening plexuses.

I may observe, that the facts I have mentioned refer only to the most ordinary description of phthisis, and that they are the result of a great number of examinations; and I contend that they prove that this form is not at all connected with other forms, and that it is erroneous to suppose that miliary is necessarily the incipient state of common tubercle. My preparations, as well as those of Mr. Quekett, show that the progress of this tubercle, from its commencement up to its perfect formation, depends, simply, upon the quantity of the deposit; for it can be seen, in some parts, occupying only a part of a cell, in others, one, two, three, or even of an indefinite number, and, in every case, to exhibit the same microscopic characters.

In this form of phthisis there appear to be two pathological states from which we can deduce an explanation of its symptoms:—first, a portion of lung is rendered impermeable to the blood; secondly, the blood is thrown upon the surrounding unaffected parts.

Whether the first of these states ought to be regarded as inflammation, or not, must ultimately remain a matter of opinion, so long as the term "*inflammation*" has no definite signification.

Without doubt, it is to the second state as a mere consequence of the first, that the attacks of hæmoptysis are due, as well as the susceptibility to severe and obstinate catarrhal symptoms. The tuberculous matter, acting like an extraneous body, will produce a constant tendency to inflammation, and, after it has been produced, prevent its removal. Hence will follow those hectic and other symptoms which result from the continued operation of any irritating body.

As regards the expectoration, this will occur most frequently from the bronchial membrane, and, most probably, is not to be distinguished from that in ordinary bronchitis. It will be only during the breaking up of a tubercle that matter truly tuberculous will be expectorated, and this, I believe, can be recognized, with certainty, by no other character than its containing fragments of the membrane of the air-cells.

P.S.—Since this paper was communicated to the Society, I have met with an instance in which tubercles existed in the lungs, liver, kidney, mesentery, and other parts,—all evidently of a scrofulous character. I injected the animal (a rabbit) with fine injection. Some parts of the lungs were studded with white masses of different sizes; others,

even as much as the third of a lobe, appeared very much like a lung which had never respired. On examining the latter, I perceived in the arterial trunks leading to those parts, distinct masses of white granular matter mixed with the injection; and, continuing the examination, I found that this appearance was due to all the capillaries being, literally, choked up with this same matter. The air-cells were free from it, and contained air. The white masses in the other parts appeared to be produced by the vessels being filled with this matter, as in the preceding, and also by its escape into the air-cells and surrounding structures. On examining the kidney, I found that the vessels were filled in the same manner as in the lungs. I mentioned this to Mr. Quekett, who told me that he had, in scrofulous cases, seen strumous matter mixed with blood, which had been pressed out from an artery going to a diseased part.

TWO CASES

OF

ANEURISM,

IN WHICH THERE WAS NEITHER PULSATION NOR ABNORMAL SOUND.

By T. A. BARKER, M.D.,

PHYSICIAN TO ST. THOMAS'S HOSPITAL.

Received June 23rd,—Read June 24th, 1845.

No medical man can have been long in practice without meeting with cases in which extensive disease of the heart and large arteries had long existed, and ultimately proved fatal, without having caused any symptoms directing the attention of the patient to the seat of disease; and there must be few experienced practitioners who, when consulted for some other complaint, have not detected organic disease of the heart, or large vessels, which had previously been overlooked by some medical attendant, not because he was incapable of detecting such disease, if he had looked for it, but because the patient had spoken of no symptoms referable to disease of those parts. Bearing in mind the frequency of such oversights, even on the part of well-informed and competent medical practitioners, I should not venture to take up the time of the So-

ciety by a history of the two following cases, if they were merely instances of aneurisms which had been overlooked during life. In the first case, it is true, a large aneurism of the arch of the aorta was not discovered until after death; and, in the second, an aneurism of the renal artery was not detected until the day before death. What makes the cases worthy of record, is, not that the aneurisms existed without giving rise to prominent and characteristic symptoms, but that, when certain symptoms had arrested my attention, leading me, in the first case, to think it highly probable, and, in the second, possible, that an aneurism existed; the most careful and frequently repeated examinations did not enable me to detect either pulsation or abnormal sounds.

Case 1.—John Moseley, aged 60, a large, muscular man, generally enjoying good health, was admitted into St. Thomas's Hospital, under my care, on the 2nd of April 1844. His illness commenced about three months before; and during that time he had been troubled with shortness of breath and cough; his voice also had changed within the same period; it was sometimes weak, sometimes shrill, and almost always hoarse. According to his own account, his symptoms had not varied, except in degree, from the time when his illness commenced, to the day of his admission into the hospital. He complained principally of cough and dyspnœa; the latter, however, seldom distressed him, unless he was making some slight exertion. The cough was frequent; caused no pain, either

in the larynx or chest; and was attended with slight frothy expectoration. The sounds of the cough and voice were such as generally accompany chronic inflammation of the larynx in an early stage. On a careful examination, however, of the fauces and larynx, neither redness, tumefaction nor tenderness could be detected. I immediately thought it possible that the cough and alteration of the voice might depend on aneurism of one of the large vessels, or on some tumour pressing upon the recurrent nerves, although the man had never complained of palpitation, or any of the ordinary symptoms of disease of the heart, or large arteries within the thorax; and I proceeded to examine the chest again, believing that aneurism might exist.

The chest was large and well formed. With the exception of a little dulness over the upper part of the sternum, it sounded well on percussion, and healthy respiratory sounds could be heard every where. Gentle percussion over the region of the heart did not indicate any increase in the size of that organ; but forcible percussion gave a dull sound over a larger space than usual. The heart's impulse was a little greater than natural; but its sounds were healthy, both as regarded their nature, degree, and the space over which they could be heard. After a careful examination of the cardiac region, I came to the conclusion that the heart was slightly hypertrophied, and that it was covered by the edge of the lung more than is usually the case. The course of the aorta, and of the large arteries

above the clavicles, was examined, but no abnormal sound could be heard, and no unusual pulsation could be felt.

The upper part of the left side of the chest, anteriorly, appeared larger than the corresponding part on the right. There was considerable tumefaction along and on each side of the course of the axillary artery, from the clavicle to the axilla. Pulsation of the prominent part could be distinctly seen, and a strong thrilling pulsation was felt when the hand was applied. No defined tumour could be detected in the pulsating part; but examination by the hand gave the impression, that the artery was greatly dilated from the clavicle to the lower edge of the pectoral muscle. On applying the stethoscope over the axillary artery, immediately below the clavicle, a loud whizzing sound could be heard at the time of each pulsation, and this sound was distinct, though much fainter, even in the axilla. The detection of disease in this part of the artery naturally increased my suspicion that there might be aneurism about the arch or innominata; and I again made a careful examination of the upper part of the chest, and above the clavicles, but without discovering any new symptom. It is true that, at times, there appeared to be a very faint systolic bruit over the left subclavian, immediately before it passes under the clavicle; but, if this sound existed at all, it was very slight, was not constant, and certainly could not be heard on, or immediately above, the sternum.

This man was examined by me several times, but

the same symptoms were always observed. On the morning of the 12th, ten days after his admission, he died suddenly whilst eating his breakfast.

Post mortem examination.—No disease was found, except in the chest. The lungs were quite healthy. The heart was much hypertrophied, and the left ventricle a little dilated: the lining membrane and valves were healthy. The examination confirmed me in my opinion that the heart had been covered by the lung, except when a complete expiration had been made.

The aorta was a little dilated at its origin, and for the first two inches of its course; a sudden and much greater dilatation then commenced, and continued to beyond the point where the left subclavian is given off. Throughout this space, the artery was at least three times its natural diameter. The dilatation, which was principally at its superior and anterior aspect, commenced and terminated abruptly. The coats of the vessel had not given way at any part, except where they were in contact with the superior portion of the sternum; the inner surface of which was ulcerated. At this point there was a small quantity of firm old coagulum, which completed the sac. There was no other coagulum within the aneurism, except such as had formed since death. Large and numerous patches of atheromatous and earthy deposit were found from the commencement of the aorta to some distance beyond the aneurism. The innominata and left carotid were dilated at their origins. The left subclavian was also dilated a little from its commencement,

to the thyroid axis, at which point it was contracted, but it dilated again immediately below the clavicle, to at least twice its natural diameter, and continued of the same size to beyond the origin of the subscapular artery. There can be little doubt that the dilatation during life, of the axillary artery, must have been much greater than it appeared after death. The loud whizzing and bruit, heard just below the clavicle, must have been caused by the contraction of the artery just above; but I was at a loss to account for the absence of bruit, and of any increased pulsation along the course of the aorta and large vessels in the neck. A mere inspection of the parts, without a knowledge of the care with which several examinations had been made during life, when I had a strong suspicion that aneurism existed, would have led me to suppose that bruit and pulsation must have been perceptible over the sternum and in the neck. I can, however, state most confidently, that if a surgeon had recommended an operation for the disease of the axillary artery, no symptom was observable between the 2nd and 9th of April which could have led to a suspicion of disease between that artery and the heart, except the peculiar sound of the voice, &c., already mentioned.

Case 2.—William Greatrex, ætat. 35, a labourer, was admitted into St. Thomas's Hospital on Tuesday, the 7th of January 1845. He was slightly emaciated, his countenance was expressive of anxiety and pain, and he lay in bed with his shoulders raised, his body bent and inclined towards the right side,

and his legs drawn up. All the account he gave of his illness was, that for eight months he had suffered much from pain in the loins and hips, which generally affected the right side only, and was always much worse on the right than on the left side. He had, however, with slight interruptions, been able to continue at his work until three weeks prior to his admission into the hospital.

When I first saw him, he complained of constant aching pain in the right lumbar region, extending along the course of the right lumbar muscles, to about four inches below Poupart's ligament, at the front and inner part of the thigh. The pain in the loins was constant, and generally very severe, though it sometimes remitted a little. It was slightly increased by any movements which called into action the lumbar muscles, and, as I have already stated, he assumed, and retained as much as possible, that position in bed which most relaxed those muscles on the right side.

When first examined by me, there was less pain than usual in the loins, but he complained so much of severe pain about the right hip and upper part of the right thigh, that I made a careful examination of the hip-joint. It was soon ascertained, however, that the pain must be caused by disease at a distance; for, not only was there no unnatural appearance about the joint and limb where the severe pain was seated, but pressure of the buttock around the joint, on the groin, and painful part of the thigh, caused no increase of pain, neither did the patient

logical considerations in favour of the non-inflammatory nature of phthisis. These facts go also to show that the obliteration of the capillaries between the air-cells is, in phthisis, produced by some force which has exerted upon them a slow and very gradual compression, such as can only be conceived to have been produced by the accumulation of tuberculous matter, in contiguous cells, pressing upon the intervening plexuses.

I may observe, that the facts I have mentioned refer only to the most ordinary description of phthisis, and that they are the result of a great number of examinations; and I contend that they prove that this form is not at all connected with other forms, and that it is erroneous to suppose that miliary is necessarily the incipient state of common tubercle. My preparations, as well as those of Mr. Quekett, show that the progress of this tubercle, from its commencement up to its perfect formation, depends, simply, upon the quantity of the deposit; for it can be seen, in some parts, occupying only a part of a cell, in others, one, two, three, or even of an indefinite number, and, in every case, to exhibit the same microscopic characters.

In this form of phthisis there appear to be two pathological states from which we can deduce an explanation of its symptoms:—first, a portion of lung is rendered impermeable to the blood; secondly, the blood is thrown upon the surrounding unaffected parts.

Whether the first of these states ought to be regarded as inflammation, or not, must ultimately remain a matter of opinion, so long as the term "*inflammation*" has no definite signification.

Without doubt, it is to the second state as a mere consequence of the first, that the attacks of hæmoptysis are due, as well as the susceptibility to severe and obstinate catarrhal symptoms. The tuberculous matter, acting like an extraneous body, will produce a constant tendency to inflammation, and, after it has been produced, prevent its removal. Hence will follow those hectic and other symptoms which result from the continued operation of any irritating body.

As regards the expectoration, this will occur most frequently from the bronchial membrane, and, most probably, is not to be distinguished from that in ordinary bronchitis. It will be only during the breaking up of a tubercle that matter truly tuberculous will be expectorated, and this, I believe, can be recognized, with certainty, by no other character than its containing fragments of the membrane of the air-cells.

P.S.—Since this paper was communicated to the Society, I have met with an instance in which tubercles existed in the lungs, liver, kidney, mesentery, and other parts,—all evidently of a scrofulous character. I injected the animal (a rabbit) with fine injection. Some parts of the lungs were studded with white masses of different sizes; others,

ilium. Half way between this latter point and the ribs, a loud systolic bruit could be heard, which gradually diminished, as the stethoscope was moved towards the ensiform cartilage, where it could scarcely be distinguished, and over the region of the heart no unnatural sound could be heard.

There could now be little, if any, doubt of the existence of aneurism of the abdominal aorta, or one of its branches, and I prophesied a speedy termination of the case, believing that a rupture of the sac had recently taken place. The man was not heard to move or complain during the night, and the next morning he was found dead in his bed.

The treatment was only palliative, and need not be detailed.

Post mortem.—The heart and lungs were healthy. The commencement and arch of the aorta exhibited a few opaque, yellow patches, but there was no disease sufficient to cause any impediment to the blood. At the commencement of the descending aorta there was a true aneurism, occupying about four inches of the artery. The dilatation was uniform throughout the whole calibre of the artery, which, about midway between the commencement and termination of the aneurism, was about twice its natural diameter. Absorption of the vertebræ, and intervertebral substance, had commenced opposite this part of the artery. Tearing the vessel downwards, numerous patches of yellow deposit were found, and these increased in size and thickness, in the neighbourhood of the large abdominal branches.

The right renal artery was much diseased in a similar manner, and there was a false aneurism of that artery, of the size, and somewhat of the shape, of a heart. The vessel had given way, almost close to its origin at its lower part. The sac was formed by the neighbouring parts and cellular membrane. A prolongation of it extended behind the pelvis of the kidney. The sac had burst, and allowed the escape of blood to such an extent into the cellular membrane and around the kidney, as to more than double the size of the original tumour. The pressure of the aneurism had caused absorption of the anterior and right lateral parts of the bodies of three lumbar vertebræ. The liver was so much enlarged as completely to cover the aneurism, and must have prevented it being felt by the hand during life, and no pulsation could have been perceived, except what might have been communicated to the liver. The enlargement of that organ was caused, in a great measure, by venous congestion:—its structure was not altered.

Looking at the aneurism, as it existed after death, it was difficult to account for the absence, at the first five examinations, of the pulsation and bruit which were so distinct the day before death took place. On carefully examining the contents of the sac, and the blood effused around it, it appeared to me highly probable,—considering the manner in which numerous large, firm, yellowish flakes of coagula were mixed with recently clotted blood, that the sac had been completely filled by layers of

dense coagula at the time when the man was first seen, so that little, if any, fluid blood passed into the sac. If these coagula were subsequently disturbed and broken down, so as to admit a small quantity of fluid blood, and the sac then burst, the difficulty of explaining the symptoms diminishes, though it is not removed, for I have several times heard a loud bellows' sound along the course of the abdominal aorta, when there was no aneurism, but only disease of its coats, not greater than was observed in the present case. I consider, therefore, that this may fairly be regarded as a case of aneurism without abnormal sound or pulsation; unless it is thought that the enlarged liver might have prevented the pulsation being felt, until the tumour had acquired increased size by the bursting of the sac. This is not my own opinion, though it may perhaps be entertained by others.

I have already alluded to the frequent existence of aneurisms, which remain undetected until after death, solely because the medical attendant had been consulted for some other disease, and had observed no prominent symptom, leading him to suspect any affection of the heart and arteries. There have been other cases, less frequent than the above, in which, after careful and repeated examination, no symptoms of aneurism were observed; but on examination after death, some cause for the absence of such symptoms was found, either in the size, shape, or situation of the aneurism, in the mode by which it was covered by other parts, or in the nature of its

communication with the artery. In neither of the cases just related, can any such explanation be given, and the care and frequency with which the examinations were made, leave no reason to suppose that any material symptoms which could have assisted me in the diagnosis were present, and overlooked. They may therefore be regarded as aneurisms of considerable size, in situations where such disease can ordinarily be detected without difficulty, but which gave rise to none of the ordinary symptoms; and looking at them in this light, I think they are not unimportant additions to our stores of pathological knowledge.

The histories of cases published, in order to excuse some glaring error of diagnosis, are generally, though often with great injustice, looked upon with suspicion, even when the author's character and professional reputation are so high as to leave no reason to suppose he can have been guilty of intentional concealment or perversion of facts. In such cases, it is often urged that the natural desire not to appear in fault may cause forgetfulness, or too low an estimate of symptoms which might with greater care and skill have led to a correct diagnosis. I am no doubt liable to be influenced by such circumstances, as well as others; but in order to give greater weight to these cases, I may remark that they cannot be related for the purpose of defending my own reputation; since they were not spoken of beyond the walls of the hospital when they occurred, and have probably been long forgotten even there.

Moreover, in both cases I expressed a strong suspicion of aneurism, from the first; whilst in one, new symptoms arising, I was able to make a correct and positive diagnosis before death took place.

14th.—The erysipelas is declining, the discoloration continuing exactly the same.

26th.—The black matter being washed off at one o'clock, Dr. Hodgkin and myself closely watched the patient, and at half-past six o'clock he was as much surprised as I was on the former occasion, on seeing the black secretion appear on the right under eyelid, in precisely the same manner as I had previously witnessed, with this difference, that to-day the discoloration was an hour later in making its appearance, than it was on the former occasion. We remained with the patient until after seven o'clock. The result of this investigation was in accordance with a statement made to me by the lady's father, a few days previously, to the effect, that the intervals between the re-appearance of the discoloration, after the removal of the black secretion, were now of longer duration than formerly.

29th.—The pulse being rather quick and hard, the patient was bled from the arm, but in consequence of the supervention of syncope, four ounces of blood were only abstracted. The blood presented no abnormal appearance, but there was great difficulty in arresting the bleeding, which rendered it necessary to re-apply the bandage three or four times.

May 1st.—The erysipelatous eruption has reappeared on the forehead and face, tongue coated with a thick white fur in the centre, bowels regular, loss of appetite; the patient has taken little nourishment the two last days; urine natural.

4th—This day there were vomited two large basins-

the black matter off, it caused her so much pain, owing to the sensitiveness of the skin, that she desisted, and, until I removed it by the application of soap and water, was not aware that it could be removed by ordinary means. The quantity of matter removed from the skin was sufficient to darken four basins of water as black as Indian ink. I was informed that about the time when this singular affection first made its appearance, Miss —— was at Belfast, under the care of Dr. Read, for a severe pain in the chest, from which she still suffers. She subsequently came to London, and was first seen by Mr. White, and afterwards by Dr. Chambers, whom she consulted a few times previous to her coming under my care. With the exception of occasional headache, and the severe pain in the chest, above mentioned, Miss —— appeared in good health; the bowels were generally confined, the catamenia regular, but scanty; her complexion fair, and the hair of a light brown colour.

Dr. A. T. Thomson saw the patient with me at this time, and as we found that the black matter could be removed from the affected parts by ablution, leaving the skin underneath of its natural appearance, we were inclined to suspect some imposition on the part of the young lady. Having made her father acquainted with our opinions, he immediately agreed to watch the case, with a view of ascertaining whether there was any ground for these suspicions. Accordingly, on the 28th of February, having myself washed off the black mat-

ter, Mr. —— and three of his friends remained with the young lady, and were present when the discoloration made its appearance on the skin, about four hours after I had removed it. Mr. —— assured me, that during the investigation he never allowed his daughter to leave his presence, and that it was utterly impossible for her to have practised any imposition on him or his friends.

Still, I confess, I was somewhat sceptical. With a view, therefore, of removing every doubt from my mind, I was requested by Mr. —— to fix a day to investigate the matter for myself, which I accordingly did on the 31st of March.

On this day (the patient having no knowledge of my visit), I washed the black matter from the right under eyelid, at one o'clock, and never allowed her to leave my presence until five o'clock, up to which hour no discoloration or alteration in the appearance of the affected part had occurred. Being now, more than ever satisfied, that the patient was imposing, I left her in the drawing-room, and proceeded as far as the door, with the intention of returning home, when her father requested me to remain until seven or eight o'clock, assuring me, that previous to that hour I should be perfectly satisfied respecting the truth of his statement, as the discoloration, according to his observations, did not appear sooner than from four to six hours after ablution. In compliance with this request, I returned to my patient, who, during the few minutes I was absent, had not left the drawing-room. Miss

—— told me that she now began to feel a pricking and burning heat in the affected parts, which invariably preceded, from about a quarter to half an hour, the appearance of the discoloration, and that she was confident the blackness would very soon show itself. At half-past five o'clock, I was astonished at seeing a small dark spot appear upon the right under eyelid, near the inner angle of the eye, which kept gradually extending towards the right temple, so that, in half an hour from its first appearance, the eyelid had become quite black, the forehead, at the same time, assuming the same intense discoloration. I remained with the patient until eight o'clock, and felt perfectly satisfied respecting the truth of the alleged phenomena.

On the 4th of April, I requested Mr. Perry to take a drawing of the case. The right half of the forehead represents the discoloration of two days' duration, and the opposite side of five, showing a very great difference in the quantity of the black matter secreted during the longer interval, when contrasted with the shorter period.*

April 8th.—The patient complained of pains in the bones, was feverish, and the tongue coated with a white fur, pulse quick; the forehead and left cheek were covered with an erysipelatous eruption, which, in the course of a few days, gradually extended down to the sternum, and along the opposite side of the neck and face.

* See Plate VI.

"Its re-actions were as follows:—

"It was insoluble in water; it yielded fatty matter to alcohol; the black colour was not changed by the action either of caustic potassa or strong nitric acid; on burning it, a strong odour of decomposing animal matter was perceived. When moistened with distilled water it reddened litmus paper. The ashes obtained by its combustion were very alkaline.

"Under the microscope was seen a confused mass apparently made up of short hairs, epithelial scales, granules, and globules of fat.

"I must remark, that the ashes obtained from the second specimen you sent me, as scraped from the face, were not so alkaline as those from the first specimen; nor did the black matter show an acid re-action. This may have been produced, however, as a difference consequent on less of the ordinary cutaneous secretion having become mixed with the carbon. The black matter vomited by the patient, when viewed under the microscope, had much the appearance of broken down animal structure. It was very acid, but contained scarcely any hydrochloric acid. It burned away easily, leaving an ash possessing an alkaline re-action. The carbon from the face was so finely divided that it burned with a vivid combustion, almost having the appearance of deflagration. Carbon is certainly the colouring principle of this black material, which, in other respects (that is, with the exception of this uncombined element being present), gives all

ful of fluid, containing an immense quantity of black matter, which subsided to the bottom of the vessels. This black matter, when examined under the microscope, as well as by analysis, was found by Dr. Rees to be identical with the specimens of black matter with which I supplied him from the patient's face; the vomited matter, and a specimen taken from the patient's face, were both examined under the microscope, by Dr. Hodgkin and myself, and found to be analogous. Both appeared and smelt exactly like soot. The vomiting lasted from the 4th to the 6th, during which period the black matter was passed by the bowels, as well as by urine, the face remaining free from any discoloration during this time. On the evening of the 6th (no matter being ejected from the stomach, or secreted from the face since the morning), the patient was seized with the most agonising pain in the stomach and bowels I ever witnessed. When called to see Miss —— I found her doubled up in bed, her head resting on her knees, and screaming out on account of the violence of the pain; 150 drops of laudanum were given, in divided doses, in the course of three or four hours, when the pain subsided, and did not return until the following evening, when it recurred again in a milder form. On the 7th, the morning after the attack of pain, the black secretion reappeared, as usual, on the forehead and face, and continued up to the 23rd of May, when it entirely ceased, having, for two or three days previous to its entire cessation, appeared in less quantity than usual.

June 17th.—Up to the present date the patient has had no return of the disease, and with the exception of occasional headache and pain in the chest, she appears in tolerably good health. It may be worthy of remark that the pain of the chest commenced about three weeks previous to the appearance of the black secretion; and notwithstanding the repeated application of leeches and blisters it still continues unmitigated. The internal treatment consisted in the administration of extract of colocynth, blue pill and James's powder, the infusion of roses and sulphate of magnesia, and occasionally the nitro-muriatic acid. Externally the decoction of oak bark with sulphate of alum was used, and to parts of the forehead a solution of the nitrate of silver, in the proportion of a scruple to an ounce of distilled water, was applied, but none of these applications had the smallest effect in checking the black secretion.

I believe there is no case on record similar to the present one; and respecting the physiological cause of the disease, I have no clear conception; but I believe the secretion to be analogous to what occurs in melanosis. The cessation of the secretion of the black matter by the cutaneous exhalants of the forehead during the period it was ejected from the stomach, bowels, and kidneys, although singular, is not unaccountable; the latter organs probably becoming a substitute for the former. The patient had no tendency whatever to hysteria. The secretion was always more abundant during the

night than the day, so much so, that when the patient washed her face at bed-time, the affected parts were invariably covered the subsequent morning with a large quantity of the black matter in a solid but moist condition.

The present case appears to me to bear no analogy to the disease designated melasma, by Linnæus, Vogel, and Plenck, which is manifestly a disease of debility dependent on a cachectic state of the constitution; neither had it any resemblance to those cases of coloured perspiration described by various authors, but more particularly by Professor Frank—"Color nunc pallide flavescens, nunc lacteus, vel croceus, sanguineus, ac interdum subviridis, cœruleus aut ater."*

I may mention, that the patient was seen by Sir B. Brodie, Dr. Bright, Dr. Roupell, and Dr. G. O. Rees, by all of whom the case was considered novel, and such as they had never before witnessed. To the last gentleman I am indebted for the subjoined analysis.

"We may regard the black matter, scraped from the face, as composed of,

Carbon.
Iron (in some unknown form of combination).
Lime.
Animal matter (albuminous).
Fatty matter.
Alkaline phosphate and chloride.

* De Curand. Homin. Morb. Epitome, tom. v., p. 27. Mannh. 1792.

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the re-actions of a common form of animal matter.

(Signed) "G. Owen Rees."

"Guildford Street, June 16th, 1845."

P.S.—Sept. 29.—Miss —— remains perfectly free from any return of the disease, and is in every respect quite well, being now free from headache and pain of the chest.

Note by the Editor.—From the unusual character of the above case, and the doubts entertained by some persons as to its genuineness, I was desired by the council to ascertain further particulars respecting it, from Dr. Read, of Belfast, who saw the case at its commencement, and also from Dr. Hodgkin, who (as is mentioned in the paper) was present on one occasion when the peculiar secretion made its appearance. In a letter which Dr. Read was so good as to favour me with, in answer to my enquiries, he states :—"The prominent object of Miss ——'s visit to me was, that she might undergo a proper examination of her chest, on account of a fixed and intractable pain somewhere about the superior mammary region, on the sternal aspect, and left side of the sternum, which had induced serious alarm, lest, at her particular age, it might be connected with a phthisical diathesis. It appeared that previous to my being consulted, there had been an outbreak of this peculiar secretion above

the brow, which had subsided, or was removed, but had just recurred, and occupied a larger surface exactly prior to her journey, and on her arrival at Belfast. Her father being an intimate friend of mine, I had the young lady removed from the hotel to my own house, for a more convenient and perfect examination of the chest, so that while with me, the malady of the skin was of subordinate or secondary importance, but I did not neglect its scrutiny. I classed it, and called it 'Pityriasis nigra,' though I had never seen that disease; a moderate magnifying power strengthened this opinion. I found that I could easily remove the black furfuraceous matter by friction, in minute laminæ or scales, leaving the subjacent cuticle very nearly normal; and I then had no doubt that the action of air and light, in a few hours, changed the exposed cuticle into the same appearance as prior to the friction and removal of the scaly surface. It is quite true it might not be a change in the cuticle itself, from the chemical action of air and light, but a palpable transudation through the exhalent vessels; in fact, a true excretion. In the note of the case I sent to London, with the patient, both suggestions were contained: the former assimilating to the name I gave the disease; the latter, conforming to the theory I proposed of its cause, which I supposed to be connected with an imperfect elimination of the uterine function."

In a second letter which I addressed to Dr. Read, I mentioned that suspicions had been enter-

tained by some, as to the genuineness of the disease, and requesting him to be so good as to inform me whether he had any doubts on the matter. Dr. Read immediately replied to the following effect:—

"I understand doubts have been entertained, and suspicions cast, on the genuineness and unsophisticated nature of the disease, from the unexampled appearance in this case, and its being unprecedented in the experience of several eminent physicians and surgeons. I saw the case very early after its commencement, and it at once attracted the most rigid observation and scrutiny I was capable of using, and the young lady, while at Belfast, resided in my own house. I have therefore no hesitation whatever in saying, that my opportunity of observation during her residence under my own eye, rendered it impossible that imposition could have been practised in this instance. I am fully conversant with the strange manœuvres occasionally practised by hysterical girls, from which no rank is exempt, but I know the father is a military gentleman of unblemished honour and truth."

From Dr. Hodgkin I received the following communication in reply to my enquiry:—

"I have perused my friend Teevan's account of his very remarkable case. It is an accurate narrative of what I either saw myself, or heard described. On the occasion alluded to, when I was present, though the coloured deposit progressively increased during upwards of half an hour (in which time I

was opposite to the young lady at dinner), absolute blackness was not produced, but the very remarkable character of the deposit, which could hardly be imitated by any artificial process, was to my mind satisfactory proof of the genuineness of the case. I noticed an extremely faint blush on the part which became subsequently discoloured."

GEO. CURSHAM, M.D.

EXPLANATION OF THE PLATES.

PLATE I.

Illustrating Mr. Erasmus Wilson's paper on the classification, structure and development of the echinococcus hominis, p. 21.—A detailed description of the figures is given at p. 35.

PLATES II. and III.

The hands of Eliza Hitchcock, illustrating Mr. Curling's case of remarkable hypertrophy of the fingers, p. 338.

Fig. 1.—Right hand.

a b c Hypertrophied fingers.

Fig. 2.—Left hand.

d e f Hypertrophied fingers.

The middle finger curved outwards by displacement of the extensor tendon.

PLATE IV.

Illustrating Dr. West's paper on cephalhæmatoma, p. 397.

Fig. 1.—Represents the exterior of the bone.

a a The anterior or coronal edge of the bone.

b The fissure in the bone.

c The edge of a section of the bone, made parallel to the sagittal suture, and showing

d The clot; effused beneath

e The dura mater.

f f The roughened appearance of the bone produced by the deposit of new bony matter.

g g The outer and posterior edge of the bone divided obliquely.

Fig. 2.—Represents the interior of the bone; from which the dura mater has been detached, though it still covers the effused blood.

a The fissure in the bone.

b b b The osseous ring, formed by the deposit of bony matter around the clot.

c The raised edge of the clot, beneath which the inner table of the skull is seen, smooth and unaltered.

d The internal layer of the dura mater, partially reflected, and showing that the deposits of bone, which are seen beneath it, had been formed between the two layers of the membrane.

PLATE V.

Illustrating Mr. Rainey's paper on the minute structure of the lungs and on pulmonary tubercle, p. 581.

Fig. 1.—Shows the abrupt termination of a bronchial tube in a bronchial intercellular passage.

a The bronchial membrane.

b The opening of an air-cell into a bronchial tube, with the capillaries surrounding it.

Fig. 2.—The course and termination of a bronchial intercellular passage.

a a Its commencement and termination.

b An air-cell opening into it.

Fig. 3.—The pulmonary membrane enclosing the capillary plexuses highly magnified.

a The plexuses.

b The free margin of the membrane, projecting beyond the vessels, and forming the immediate boundary of the opening of one air-cell into another.

Fig. 4.—The tubercular matter situated in the air-cells with the remains of the capillary plexuses between the cells.

a The tubercular matter.

PLATE VI.

Represents the black secretion from the skin of the forehead and upper part of the face in the case related by Mr. Teevan, p. 611. The lighter hue on the right half of the forehead is only of two days' duration; the darker and thicker coating on the left, is of five.

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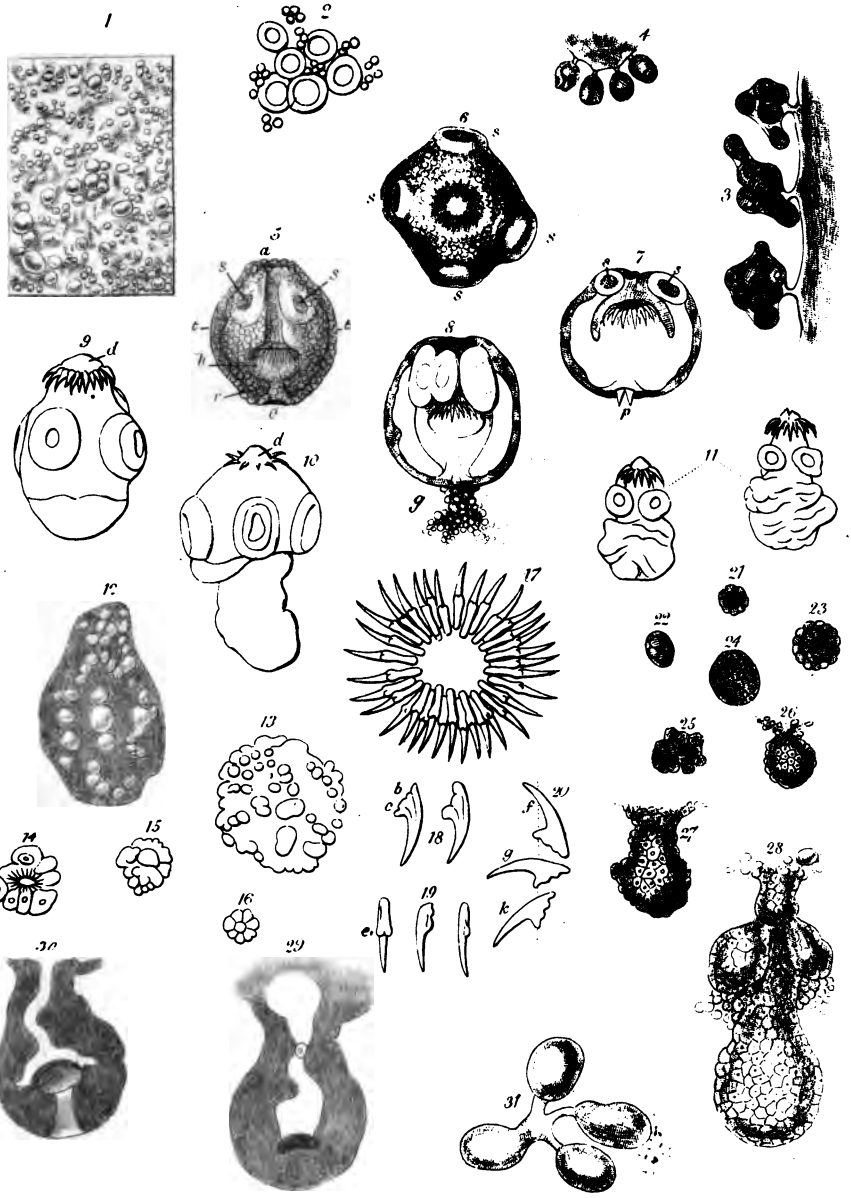

Erasmus Wilson del.

W. Sowerby jr.

a

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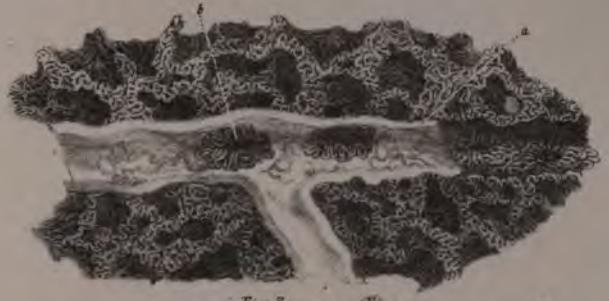

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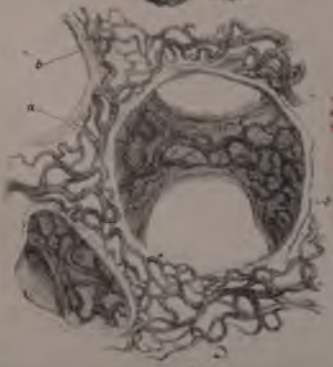

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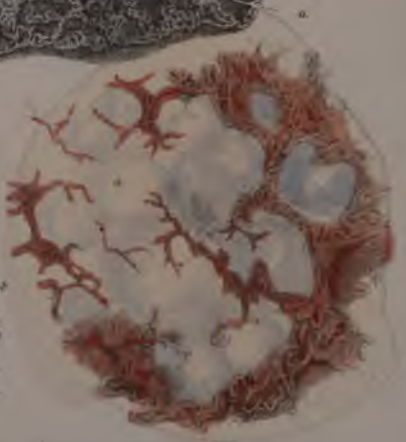

Fig 4

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